PB 188 073

AIR POLLUTION ASPECTS OF PHOSPHORUS AND ITS COMPOUNDS

Yanis C. Athanassiadis

Litton Systems, Incorporated
Bethesda, Maryland

September 1969

CLEARINGHOUSE
FOR FEDERAL SCIENTIFIC AND TECHNICAL INFORMATION

Distributed . . . 'to foster, serve and promote the nation's economic development and technological advancement.'

U.S. DEPARTMENT OF COMMERCE/National Bureau of Standards

This document has been approved for public release and sale.

PB188073

TECHNICAL REPORT

Reproduced by the
CLEARINGHOUSE
for Federal Scientific & Technical
Information Springfield Va. 22151

CONTINENTAL SYSTEMS, A Division of [illegible], 700 PLANT STREET, [illegible]

AIR POLLUTION ASPECTS

OF

PHOSPHORUS AND ITS COMPOUNDS

Prepared for the
National Air Pollution Control Administration
Consumer Protection & Environmental Health Service
Department of Health, Education, and Welfare
(Contract No. PH-22-68-25)

Compiled by Yanis C. Athanassiadis

Litton Systems, Inc.
Environmental Systems Division
7300 Pearl Street
Bethesda, Maryland 20014

September 1969

FOREWORD

As the concern for air quality grows, so does the concern over the less ubiquitous but potentially harmful contaminants that are in our atmosphere. Thirty such pollutants have been identified, and available information has been summarized in a series of reports describing their sources, distribution, effects, and control technology for their abatement.

A total of 27 reports have been prepared covering the 30 pollutants. These reports were developed under contract for the National Air Pollution Control Administration (NAPCA) by Litton Systems, Inc. The complete listing is as follows:

Aeroallergens (pollens)
Aldehydes (includes acrolein and formaldehyde)
Ammonia
Arsenic and Its Compounds
Asbestos
Barium and Its Compounds
Beryllium and Its Compounds
Biological Aerosols (microorganisms)
Boron and Its Compounds
Cadmium and Its Compounds
Chlorine Gas
Chromium and Its Compounds (includes chromic acid)
Ethylene
Hydrochloric Acid
Hydrogen Sulfide
Iron and Its Compounds
Manganese and Its Compounds
Mercury and Its Compounds
Nickel and Its Compounds
Odorous Compounds
Organic Carcinogens
Pesticides
Phosphorus and Its Compounds
Radioactive Substances
Selenium and Its Compounds
Vanadium and Its Compounds
Zinc and Its Compounds

These reports represent current state-of-the-art literature reviews supplemented by discussions with selected knowledgeable individuals both within and outside the Federal Government. They do not however presume to be a synthesis of available information but rather a summary without an attempt to interpret or reconcile conflicting data. The reports are

necessarily limited in their discussion of health effects for some pollutants to descriptions of occupational health exposures and animal laboratory studies since only a few epidemiologic studies were available.

Initially these reports were generally intended as internal documents within NAPCA to provide a basis for sound decision-making on program guidance for future research activities and to allow ranking of future activities relating to the development of criteria and control technology documents. However, it is apparent that these reports may also be of significant value to many others in air pollution control, such as State or local air pollution control officials, as a library of information on which to base informed decisions on pollutants to be controlled in their geographic areas. Additionally, these reports may stimulate scientific investigators to pursue research in needed areas. They also provide for the interested citizen readily available information about a given pollutant. Therefore, they are being given wide distribution with the assumption that they will be used with full knowledge of their value and limitations.

This series of reports was compiled and prepared by the Litton personnel listed below:

Ralph J. Sullivan
Quade R. Stahl, Ph.D.
Norman L. Durocher
Yanis C. Athanassiadis
Sydney Miner
Harold Finkelstein, Ph.D.
Douglas A. Olsen, Ph.D.
James L. Haynes

The NAPCA project officer for the contract was Ronald C. Campbell, assisted by Dr. Emanuel Landau and Gerald Chapman.

Appreciation is expressed to the many individuals both outside and within NAPCA who provided information and reviewed draft copies of these reports. Appreciation is also expressed to the NAPCA Office of Technical Information and Publications for their support in providing a significant portion of the technical literature.

ABSTRACT

High concentrations of phosphorus and its compounds, especially yellow elemental phosphorus, may cause adverse physiological effects ranging from skin irritation to systemic poisoning. In very low concentrations, many of the organic and some of the inorganic compounds of phosphorus inhibit basic metabolic processes in mammals, especially the enzymatic activity of cholinesterases.

In vegetation, high concentrations of phosphorus induce a deficiency of zinc, copper, and molybdenum, resulting in reduced growth.

Certain phosphorus compounds—for example, phosphoric acid—are also quite corrosive to materials.

The major sources of phosphorus air pollution are the industries engaged in the production of phosphate fertilizers, phosphoric acid, phosphorus pentoxide, and phosphorus chemicals for industrial uses. The application of organophosphorus pesticides may also result in environmental air pollution. This is discussed in the companion report, "Air Pollution Aspects of Pesticides."

Recently, various organic phosphorus compounds have been used as combustion chamber deposit modifiers as well as corrosion inhibitors for motor gasolines and aviation fuels. Thus there is a possibility that automobile emissions may contain toxic phosphorus compounds.

Phosphorus concentrations in the ambient air of Los Angeles (1954) and Cincinnati (1966) were found to average 1.43 $\mu g/m^3$, and 0.22 $\mu g/m^3$, respectively. Measurements on national or regional scales are not available.

No information has been found on economic costs of phosphorus air pollution. Investment in air pollution control equipment within the phosphate industry has been relatively high, especially in Florida, where the phosphate fertilizer industry is concentrated. Operating costs of control equipment in this section of the fertilizer industry have been estimated at $6 million annually; this also includes the control of fluoride emissions.

Most of the abatement systems used in the phosphorus industry are not specific for phosphorus, since often the major pollutant is fluoride emissions (in the phosphate fertilizer industry). Those systems which are used include combinations of scrubbers; cyclones; mist eliminators; high-energy, wire-mesh contactors; and electrostatic precipitators.

Methods available for quantitative analysis of phosphorus in the atmosphere include colorimetric (molybdenum blue method), fluorescent, and spectrophotometric methods. Some of these methods permit determinations down to the microgram level.

CONTENTS: Phosphorus and phosphorus compounds

FOREWORD

ABSTRACT

1. INTRODUCTION 1

2. EFFECTS 3

2.1 Effects on Humans 3
2.1.1 Elemental Phosphorus 4
2.1.2 Inorganic Compounds 5
2.1.3 Organic Compounds 7
2.2 Effects on Animals 11
2.2.1 Commercial and Domestic Animals 11
2.2.2 Experimental Animals 12
2.2.2.1 Inorganic Compounds 12
2.2.2.2 Organic Compounds 14
2.3 Effects on Plants 15
2.4 Effects on Materials 18
2.5 Environmental Air Standards 18

3. SOURCES 20

3.1 Natural Occurrence 20
3.2 Production Sources 20
3.3 Product Sources 23
3.3.1 Phosphoric Acids 23
3.3.2 Phosphate Fertilizers 24
3.3.3 Phosphine 25
3.4 Other Sources 26
3.4.1 Oil-Fired Boilers 26
3.4.2 Iron and Steel Industry 26
3.4.3 Transportation Sources 27
3.5 Environmental Air Concentrations 28

4. ABATEMENT 30

5. ECONOMICS 32

6. METHODS OF ANALYSIS 34

6.1 Sampling Methods 34
6.1.1 Free Phosphorus in Air 34
6.1.2 Phosphoric Acid (Plants, Stack Gasses) . 34
6.1.3 Phosphine 35

CONTENTS (Continued)

6.2 Quantitative Methods 35
6.2.1 Phosphorus and Phosphoric Acid 35
6.2.2 Phosphine 37
6.2.3 Organophosphorus Pesticides 37

7. SUMMARY AND CONCLUSIONS 38

REFERENCES

APPENDIX

LIST OF FIGURES

1. Marketed Production, Apparent Consumption, and Exports of Phosphate Rock, 1900-66 49

2. Flow Diagram of Phosphate Rock Storage and Grinding Facilities 50

3. Flow Diagram of Process for Production of Superphosphoric Acid 51

4. Flow Diagram of Wet Process Phosphoric Acid Plant 52

5. Flow Diagram of Furnace Phosphoric Acid Plant . . . 53

6. Flow Diagram: Production of Run of Pile and Granular Triple Superphosphate 54

7. Flow Diagram of Normal Superphosphate Plant 55

8. Cumulative Particle Size Distribution of Phosphate in Cincinnati and Fairfax, Ohio 56

LIST OF TABLES

1. Effects of Excessive Application of Phosphate on Sour-Orange Seedlings 16

2. Threshold Limit Values for Phosphorus and Certain Compounds 19

3. Emissions and Abatement Devices Used in Seven Phosphate Fertilizer Plants 31

4. Properties, Toxicity, and Uses of Phosphorus and Some Phosphorus Compounds 57

5. Comparative Acute Toxicity of Cholinergic Organophosphous Insecticides in Normal Male and Female Rats 68

6. Comparative Toxicities of Some Phosphorus Compounds for Experimental Animals 69

7. Production of Phosphorus Chemicals 72

8. Growth of Phosphoric Acid Industry in United States . . 73

9. Thermal-Process Phosphoric Acid Establishments in United States 74

1. INTRODUCTION

Phosphorus and its compounds have varying effects depending upon the chemistry and concentration. For example, yellow (white) phosphorus is a protoplasmic poison, while red phosphorus is not; and some of the organic and inorganic compounds of the element are comparatively nontoxic even at the gram level, while some others are highly toxic in concentrations at the microgram level. Very few of the known phosphorus compounds have been investigated with respect to their toxic effects.

The adverse effects on humans and animals exposed to concentrations of phosphorus vary from a slow, cumulative and mostly irreversible inhibition of cholinesterase activity to death from poisoning, depending basically on the physicochemical form of the elements and its concentration. The human nervous system is the most affected by high concentrations of phosphorus.

The compounds known to be emitted in appreciable quantities into the ambient air are phosphorus oxides, phosphoric acid, and the organophosphorus compounds, found mostly in agricultural chemicals. Other organic phosphorus compounds are very probably emitted into the ambient air by the chemical industry from processes in which phosphorus products are intermediate and/or final outputs.

The potential of phosphorus and its compounds as air pollutants may possibly be increasing, since production of phosphate rock has increased by more than 200 percent since 1950. This may pose a pollution problem in the areas where industrial emission sources are concentrated. Also, the use of phosphorus compounds as additives in gasoline and aviation fuels may pose a problem.

In this report the phosphorus compounds will be divided into two main groups for discussion--the inorganic phosphorus compounds and the organic phosphorus compounds.

2. EFFECTS

Elemental phosphorus (yellow) is a protoplasmic poison. Some of its compounds, especially organic phosphates, can also be lethal to man and animals in the case of exposure to high air concentrations. Exposure to relatively low concentrations can produce serious disturbances of basic metabolic processes. Acute or chronic effects from ambient air concentrations have been studied only with respect to organophosphorus pesticides. Such studies are few and do not provide data on concentrations; however, they have established that the symptoms observed are specific for and quite similar to those found in experimental studies on the toxicity of organophosphorus compounds.

2.1 Effects on Humans

The adverse effects of human exposure to high concentrations of phosphorus and its compounds vary widely both in intensity and type, depending on the physicochemical form of phosphorus. Some of the organic compounds of phosphorus are known to be harmless to humans, while others have been shown to be highly toxic. The known adverse bioeffects of phosphates at the molecular level include inhibition of a number of important enzymatic activities. The properties, toxicity, and uses of phosphorus and certain of its compounds are given in Table 4 in the Appendix.

2.1.1 Elemental Phosphorus

There are three different types of elemental phosphorus: yellow (white), red, and black; each has substantially different physicochemical and biological properties, some of which are not yet well understood.

Elemental yellow phosphorus is an extremely toxic substance considered to be a protoplasmic poison. Its toxic effect is apparently due to its potent reducing properties, which cause disturbances of intracellular oxidation processess.[47] Chronic exposure to small quantities of phosphorus causes epithelial damage of the bone capillaries with thrombosis and bone necrosis.[47]

The heart is one of the organs affected by phosphorus, and several electrocardiographic anomalies have been reported after exposure to phosphorus.[12,14,15,50] In a total of 115 patients who attempted suicide with "electric paste" (rat poison containing 2.5 percent phosphorus), 35 (30 percent) died, mostly from acute cardiovascular collapse.[14,15]

Red phosphorus is considered comparatively harmless; however, inhalation of its dust has been reported to cause pneumonia.[47]

Black phosphorus is an allotropic form that is synthesized under high laboratory pressures and is not found under normal conditions of temperature or pressure.

2.1.2 Inorganic Compounds

Phosphine (PH_3) is a very toxic gas which ignites spontaneously in air. This gas produces chronic effects similar to that observed with phosphorus.[52] Acute effects of phosphine inhalation include dyspnea, weakness, vertigo, bronchitis, edema, convulsions, and death. The odor threshold is 2 ppm (2.6 $\mu g/m^3$ by volume).[29] Phosphine acts on the central nervous system and the blood. A concentration of 2,000 ppm (2,860,000 $\mu g/m^3$) is lethal to man in a few minutes. Concentrations from 400 to 600 ppm (572,000 to 878,000 $\mu g/m^3$) are dangerous to life after exposures of 30 to 60 minutes. The maximum concentration tolerated for a 1-hour exposure is in the range of 100 to 190 ppm (143,000 to 271,000 $\mu g/m^3$), and the maximum that can be tolerated for several hours without appearance of symptoms is 7 ppm (10,010 $\mu g/m^3$).[27]

Phosphorus trichloride (PCl_3) enters the body as a vapor through the respiratory tract, causing suffocation, bronchitis, edema, and inflammation of the lungs. The vapors of PCl_3 are irritating to skin, mucous membranes, and the respiratory tract.[27,31,51] Exposure to the vapors may result in cough, painful inflammation of the eyes, burning in nose and throat, bronchial catarrh, dyspnea, and after severe exposure, death.[23,52,62] A concentration of 600 ppm (3,600,000 $\mu g/m^3$) is lethal within a few minutes.

Concentrations of 50 to 80 ppm (300,000 to 480,000 $\mu g/m^3$) are injurious after an exposure of 30 to 60 minutes. The range of concentrations from 2 to 4 ppm (12,000 to 24,000 $\mu g/m^3$) can be tolerated for exposures of as long as 60 minutes without serious symptoms.[27]

Phosphorus pentachloride (PCl_5) is also poisonous, causing adverse physiological effects similar to those described for phosphorus trichloride.[27]

Phosphorus oxychloride ($POCl_3$) is also poisonous and causes symptoms similar to those described for phosphorus trichloride.[27] In the same manner, the vapors of $POCl_3$ are irritating to skin, mucous membrances, and the respiratory tract.[26,28] Exposure to vapors may again result in cough, painful inflammation of the eyes, burning in nose and throat, bronchial catarrh, dyspnea, and after severe exposure, death.[23,52,62]

Zinc phosphide (Zn_3P_2), which is extremely toxic, may give off phosphine and accidentally cause fatalities.[47]

Tricalcium phosphate ($Ca_3(PO_4)_2$) is used in the production of some dentifrices and may cause a problem because of its low solubility (0.002 percent). Crystals of tricalcium phosphate were found in the viscera, lymph nodes, and enteric mucosa of a patient who died from carcinomatosis. The presence of these crystals was explained as the result

of excessive and careless use of a "shake-out" denture cleaner powder. Most of the particles of this powder were smaller than 5 μ* and probably were inhaled. To test the hypothesis that tricalcium phosphate crystals were associated with the observed systemic lesions and cancer, experiments were conducted with white rats[63] (see Section 2.2).

Inhalation of the dust of calcium phosphate rock has been found to affect the respiratory system adversely. Comparison of the ventilatory capacity of 18 men before and after exposure to dust revealed that there was a significant decrease in the forced expiratory volume after exposure to the dust over several hours; greater and more consistent decreases were observed in those men who had a history of persistent cough accompanied by sputum.[21] Phosphate rock dust may also be emitted in significant quantities from fertilizer manufacturing plants.

2.1.3 Organic Compounds

The effects of exposure to organic phosphorus compounds through inhalation vary from acute systemic poisoning to slow and cumulative inhibition of basic metabolic processes. The most well-studied effect is the inhibition of cholinesterase activity. Cholinesterase (specific, true, or acetylcholinesterase) is an enzyme located in most tissues, especially conductive tissues (brain and nerves), erythrocytes, skeletal

---

*μ=micron.

muscles, adrenal and salivary glands, lungs, liver, and stomach. The enzyme hydrolyzes acetycholine to acetate plus choline, and in addition, splits other choline esters. Organophosphorus compounds also inhibit pseudocholinesterases (nonspecific), and some compounds are effective in extremely low concentrations. For example, tetraethyl pyrophosphate is an effective inhibitor at a molar concentration of 10^{-16}, and this effect appears to be irreversible.[37]

Analysis of the enzymatic action of cholinesterase has shown that when the enzyme reacts with alkylphosphate, it becomes phosphorylated. The result is a phosphorylated enzyme which, in contrast to cholinesterase, does not react with water. Since its active side is thus blocked, it becomes deactivated.[27]

The inhibition of cholinesterase by alkylphosphates may lead to accumulation of acetylcholine in synaptic junctions and thus lead to hypersensitivity. Acetylcholine also accumulates at the endings of the postganglionic cholinergic nerves; at muscular end-plates; and in the brain. The toxicity in man and warm-blooded animals is extraordinarily high. The acetylcholine at the end-plates of skeletal muscles leads to muscular weakness and possibly tonic-clonic convulsions. In acute poisoning, cholinesterase is distinctly reduced (by more than 20 percent) but falls more rapidly in the plasma than in the erythrocytes. In chronic poisoning, the determination

of erythrocyte cholinesterase is considered a more reliable indicator than the determination of plasma cholinesterase.[48]

A follow-up study in California[72] (1963-1964) investigated the cases of 235 individuals who reportedly were occupationally exposed to organic phosphates in 1960. About two-thirds of these cases occurred in agricultural workers, and the remaining third among workers in chemical companies, State and local governments, nurseries, packing houses, and pest control operations. The study was based on personal interviews by a physician, limited physical examination, and laboratory measurements, including determination of blood cholinesterase activity in two 5-ml samples of blood and in a number of samples, measurements of serum glutamine oxaloacetic transaminase, serum glutamic pyruvic transaminase, thymol turbidity, and packed red blood cell volumes (hematocrits). The effects of acute, systemic insecticide poisoning varied, and the cases were divided into the following categories:

In Group 1 (87 cases), the symptoms included effects of parasympathetic stimulation such as headache, nausea, weakness, chest pain, vertigo, vomiting, nervousness, sweating, disturbed vision, shortness of breath, and excessive salivation. These symptoms were confirmed by depressed blood cholinesterase levels.

In Group 2 (91 cases) the above described symptoms of parasympathetic stimulation were also observed, but without laboratory confirmation.

In Group 3 (57 cases), no information on symptoms and laboratory findings was provided. However, organic phosphate poisoning was reported.

Of the total 235 cases originally reported, only 114 individuals stricken with acute organic phosphate poisoning were located and interviewed. The insecticides implicated were as follows:

Parathion	36 cases
Phosdrin	30 cases
Thimet	12 cases
Systox	5 cases
TEPP	2 cases
Trithion	2 cases
Malathion	3 cases
Phosphamidon	1 case
Combinations	23 cases

Exposure to these compounds varied. The majority of persons studied (74.6 percent) were directly exposed through mixing and loading of aerial sprays, ground spraying, and work in chemical plants. Another group (22.8 percent) was indirectly exposed as a result of picking fruits and vetetables, planting, irrigating, cultivating, and walking through the fields. The remainder (2.6 percent) were exposed by incidental contact. For the 114 cases traced, 72 blood analyses were made, and 57 of these showed low cholinesterase activity. Of the 114 individuals, 20 could no longer tolerate smelling or contact with insecticides: 12 persons experienced nausea and/or vomiting after even a whiff of these substances, and five others developed headache. Many individuals had avoided reexposure by shifting to other work.

However, two-thirds returned to work, and 10 of these experienced subsequent attacks of acute poisoning.

A study[30] was made of a group of patients with chronic tricresyl orthophosphate ($C_{21}H_{21}O_4P$) poisoning who had been taking this organophosphorus drug orally in doses of 200 μg. Six of the patients showed symptoms of central and peripheral nervous system damage in the form of a psychoorganic syndrome accompanied by sexual and various nervous disorders. The symptoms of mental disorders were at onset similar to those of neurasthenia; however, the full clinical picture developed only after 4 years. Sexual disorders consisted of an increase in libido, which gradually developed into complete impotence.

More detailed information on the effects of organophosphate compounds can be found in the companion report on pesticides.

2.2 Effects on Animals

2.2.1 Commercial and Domestic Animals

Einbrodt[18] studied the effects on horses of mine dust containing phosphorus pentoxide. Analysis of the lungs of three mine horses (in Germany) showed average concentrations of phosphorus pentoxide amounting to about 20 percent of the total dust content of the lungs. The highest concentration of phosphorus pentoxide was found in the bronchi (23 to 29 percent).[18]

2.2.2 Experimental Animals

2.2.2.1 Inorganic Compounds

Phosphorus trichloride (PCl_3) hydrolyzes rather rapidly, producing hydrogen chloride and phosphorus acid (H_3PO_3). When the hydrolysis takes place in the presence of moist air, fumes are produced.

Experiments in which 10 rats and 10 guinea pigs inhaled dispersed PCl_3 vapors at concentrations of 300,000 to 600,000 $\mu g/m^3$ (total particulate) gave the following results. During exposure, the animals were restless and nervous, and porphyrin secretions developed around their eyes. The nostrils and paws were swollen and edematous shortly after exposure and necrosis was observed in the nasal epithelium and its substructure. The major histological alterations observed consisted of nephrosis of the kidney tubules in the corticomedullary region. All animals died within a 10-day period after exposure. In the case of animals exposed to ammoniated PCl_3 at concentrations of 1,100,000 to 2,500,000 $\mu g/m^3$, some animals died within 3 days after exposure. All animals developed severe pulmonary edema while the lungs of the animals which survived exposure showed extensive areas of fibrosis in the alveolar walls.[79]

The hydrolysis of phosphorus oxychloride ($POCl_3$) occurs rapidly in water, and quickly enough on contact with moist air to cause fuming. The hydrolysis results in the formation of hydrogen chloride (HCl) and phosphoric acid (H_3PO_4).[79]

Other experiments in which 10 rats and 10 guinea pigs inhaled $POCl_3$ vapors at concentrations of 30,000 $\mu g/m^3$ (total particulate) gave the following results. During exposure, the animals were restless and nervous, and porphyrin secretions developed around the eyes. All deaths occurred within 48 hours after exposure. The signs of toxicity gradually abated in surviving animals and disappeared by the end of the 14-day experimental period. The lungs of the dead animals were dark red and showed plugs in the lumen of bronchi and bronchioles, while the alveolar spaces around the lumen plugs were edematous and hemorrhagic. Animals exposed to aerosols from the dispersion of ammonia-neutralized $POCl_3$ showed the same pattern of survival times and histological changes as the animals exposed to pure vapors of $POCl_3$.

Tricalcium phosphate, of the type and particle size (0.1 to 5 μ) contained in some toothpastes, was fed to 12 white rats for periods of 3 days to 7 weeks; another 21 rats were injected with 50,000 μg of a denture cleanser that contained tricalcium phosphate. Examinations showed that crystals of the phosphate had entered the intestinal mucosa of the rats after ingestion and the lungs after injection. These crystals were deposited in tissues and caused mild or severe inflammation and granulomas.[57] However, the results of these experiments were inconclusive with respect to a possible causal relation between cancer and the tricalcium phosphate crystals.[57]

2.2.2.2 Organic Compounds

The toxicity of the organic phosphorus derivatives to mammals is directly proportional to the inhibition of acetylcholinesterase produced. The phosphorus insecticides act by deactivating (phosphorylating) the enzymes they attack. A lowering of mammalian toxicity was achieved in recent years by using pesticides which are toxic to insects but not to mammals. (See companion report on Pesticides.)

The comparative toxicity of a number of organophosphorus insecticides is given in Table 5 in the Appendix.

Experiments with mice exposed to inhalation of ethylomercuric phosphate at concentrations equivalent to 0.03 to 0.04 $\mu g/m^3$ of mercury was fatal to some mice after the 3rd and 4th hour of exposure. Concentrations of ethylomercuric phosphate above 0.04 $\mu g/m^3$ killed the mice within 1 hour, while 0.03 to 0.04 $\mu g/m^3$ of metallic mercury failed to cause death of the experimental animals even after 8 hours exposure.[77]

Mice died 6 to 15 hours after exposure to ethylomercuric phosphate vapor at concentrations equivalent to 0.006 to 0.009 $\mu g/m^3$ of mercury, while mice exposed to identical concentrations of metallic mercury vapor did not die.[77] (See companion report on Mercury.)

Methyl phosphonic dichloride hydrolyzes rapidly enough in the presence of moist air to produce fumes. The products of its hydrolysis are hydrogen chloride and

methyl phosphonic acid ($CH_3PO(OH)_2$).[69] The vapors are irritating to the skin, the mucous membranes, and the respiratory tract.[26,28,51]

Experiments with rats which inhaled vapors of CH_3POCl_3 for a 4-hour period at an average concentration of 360,000 $\mu g/m^3$ showed the following results. During exposure the animals were restless and nervous, and the skin around the eyes was swollen with porphyrin secretions. Breathing became slow and difficult, necrosis was observed on paws and nostrils, and the stomach was distended. In addition to necrotic tissues, there were hemorrhagic edemata, areas of ulceration in the stomach walls, hemorrhage in the pancreas and thymus, tubular nephrosis, and excessive fat deposits in the liver, kidney, heart, and skeletal muscles. In rats exposed to ammonia-neutralized CH_3POCl_2 at a concentration of 3,600,000 $\mu g/m^3$, hemorrhagic edema was observed in the lungs, and death occurred within a day after exposure.[79] Comparative toxicities for experimental animals of certain phosphorus compounds are presented in Table 6 in the Appendix.

2.3 Effects on Plants

Phosphorus is an essential element of plant tissues, but excessive concentrations produce adverse effects. Most of these types of effects result from antagonism among trace elements. The majority of the studies have been concerned with excessive applications of phosphates in fertilizers.

Heavy applications of phosphates can induce marked deficiencies of trace elements—especially zinc and copper—as shown in Table 1.

TABLE 1

EFFECTS OF EXCESSIVE APPLICATION OF PHOSPHATE ON SOUR-ORANGE SEEDLINGS[6]

Phosphate (lb/per acre)	Shoot Weight (grams)	Leaf Composition (ppm)	
		Copper	Zinc
0	31	5.3	28
76	34	4.5	30
360	26	2.0	20
900	22	1.0	21
1,800	14	1.4	12

Phosphate-induced zinc deficiency is very common in greenhouse crops, such as tomatoes. It has also been demonstrated that adding phosphates to the soil where citrus plants are grown results in decreased absorption of boron, copper, and zinc, even though the phosphates decrease the soil pH.[6,7,10,35,44] Moreover, the induced acute copper deficiency is not influenced by the type of soil. In acid soils, the uptake of zinc and boron is usually retarded, while in alkaline soils excess phosphate decreases molybdenum uptake. Application of heavy doses of superphosphate result in a marked decrease in the zinc content of oats[58] and flax.[35,40] Heavy applications of phosphate in certain soils may increase the molybdenum content of the vegetation as much as tenfold.[69]

In some parts of Great Britain, where the copper dificiency in cattle is largely induced by high levels of molybdenum in the diet, excessive concentrations of phosphates in pastures contribute to copper deficiency by both decreasing the available copper and increasing the molybdenum content of the grass.

Even though the adverse effects of excess phosphates are usually indirect, they are nevertheless serious enough to be taken into account. For example, phosphate-induced zinc deficiencies are very extensive, being found in this country in Wisconsin, throughout the Cotton Belt, and on the Pacific Coast.[5,74,75] The extent to which industrial sources of phosphorus air pollution contribute to the excessive concentration of phosphates in soils is not known.

Two recent studies have shown that some phosphorylated compounds—mainly sugar phosphates—inhibit carbon dioxide fixation by inert spinach chloroplasts,[3] and that addition of phosphorus increases the severity of spinach injury produced by fumigation with ozonated hexene.[8]

Studies of the effect of fluoride on the succinic oxidase system in cauliflower mitochondria showed that in the presence of phosphate, fluoride caused marked inhibition of the succinic 2,6-dichlorophenolindophenol reductase. It is believed that this inhibition results from the formation of an enzyme-fluorophosphate complex. An oxidative phosphorylation were inhibited. It has also been shown that concentra-

tions of phosphate in plant tissue of 0.001 μg and greater definitely inhibit the acitivity of succinic dehydrogenase, and that inhibition increases with higher concentrations.[36]

2.4 Effects on Materials

Very limited information has been found regarding adverse effects of ambient air concentrations of phosphorus and its compounds on materials. It is known that phosphorus pentabromide, pentachloride, and pentoxide, are corrosive, as well as phosphorus tribromide.[38] Phosphoric acid (in solution) is very corrosive to ordinary ferrous metals and alloys, particularly at temperatures above 85° C.[54] Wet phosphoric acid is much more corrosive to metals than is phosphoric acid made from elemental phosphorus because of the impurities (including fluoride) contained in the wet-process acid.

2.5 Environmental Air Standards

The threshold limit values adopted and recommended in 1967 by the American Conference of Governmental Industrial Hygienists for phosphorus and some of its compounds are shown in Table 2.

The hygienic norms established in U.S.S.R. for ground-level concentrations in the ambient air of phosphorus pentoxide are as follows:[61]

Single maximum concentration: 150 $\mu g/m^3$

Average 24-hour concentration: 50 $\mu g/m^3$

TABLE 2

THRESHOLD LIMIT VALUES FOR PHOSPHORUS AND CERTAIN COMPOUNDS[60]

Substance	Threshold Limit Value (ppm)	Threshold Limit Value ($\mu g/m^3$)
Phosdrin (Mevinphos)--skin*		100
Phosphine	0.3	400
Phosphoric acid		1,000
Phosphorus (yellow)		100
Phosphorus pentachloride		1,000
Phosphorus pentasulfide		1,000
Phosphorus trichloride	0.5	3,000

*Potential contribution to the overall exposure by the cutaneous route, including mucous membranes and eye, either by airborne or direct contact with the substance.

3. SOURCES

3.1 Natural Occurrence

Natural phosphates are divided into three classes on the basis of the metal or metals to which the phosphorus is bound. The three major classes are aluminum (iron) phosphates, calcium-aluminum (iron) phosphates, and calcium phosphates. The last class is the most important one for the phosphate fertilizer industry; its principal members are bone and apatite found in phosphate rock. Since the discovery in 1867 of deposits in South Carolina, phosphate rock has become increasingly important and is now practically the only source of Phosphorus. The average concentrations of phosphorus pentoxide (P_2O_5) were estimated (1961) as 33.4 percent in phosphate rock from the mines of Florida (land pebbles), 32.0 percent in that from Tennessee, and 31.2 percent in phosphate rock from the Western States. The major constituents of phosphate rock are P_2O_5 (27.5 to 33.0 percent); calcium oxide (41.9 to 46.1 percent); volatile matter (1.9 to 7.0 percent); fluorine (2.9 to 4.4 percent); carbon dioxide; and oxides of silica, iron, aluminum, sulfur, potassium, and magnesium.[71]

3.2 Production Sources[41-45,71]

The major portion of all phosphorus produced comes from phosphate rock. Domestic marketable production of phosphate rock displays a very fast rate of growth, as shown in Table 7 and Figure 1, Appendix. Starting with less than 2 million

long tons in 1900, marketable production reached 4 million tons in 1940, 10 million tons in 1950, approached 20 million tons in 1960, and reached 35 million tons in 1966. A total of 100 million long tons of rock with a phosphorus pentoxide (P_2O_5) content of 16.43 percent was mined in 1966.

In 1965, about 73 percent of the total domestic output was produced in Florida, 10 percent in Tennessee, and most of the remaining 17 percent in Idaho, Montana, Arkansas, Utah, and Wyoming, in the order listed. Florida's phosphate rock contains from 20 to 35 percent phosphorus pentoxide.

Most phosphate rock is used for the production of fertilizers. The distribution of phosphate rock sold or used by producers during the period 1965-1967 was as follows:

	Percent		
	1965[44]	1966[45]	1967[46]
Phosphoric acid (wet process)	21.0	28.5	41.0
Triple superphosphate	21.5	27.6	16.0
Electric furnace phosphorus	29.0	23.7	21.5
Oridnary superphosphate	25.0	17.9	18.3
Other (mostly for agriculture)	3.5	2.3	3.2

Emission sources of particulates in storing and grinding phosphate rock are shown in Figure 2 in the Appendix.

Crude phosphates and phosphate fertilizers imported into the United States represent less than 1 percent of the total domestic consumption. Thus in 1966, the imports of crude phosphates amounted to 178 short tons or about 0.7 percent of the domestic apparent consumption, while imports of phosphate fertilizers and fertilizer materials amounted to 268 short

tons. Most of the small amounts of phosphate rock imported is used as animal feed supplement because of the rock's low fluorine content.[45]

Elemental phosphorus is produced as a final product for the chemical industry and as an intermediate product in the production of phosphoric acid and fertilizers.

Analysis of the flue dust samples obtained from the phosphorus furnace operations of the Monsanto Chemical Company (Monsanto, Tenn.) showed that phosphorus pentoxide (P_2O_5) as particulate constituted 18.89 percent of the flue dust. Data on gases evolved from the flue dust during heating showed that phosphine and phosphorus pentoxide fumes (collected in water) amount to 0.09 and 0.67 percent by weight of the total charge.[78] Flue dust is carried by the furnace gases along with the vaporized elemental phophorus and is collected, while hot, by an electrostatic precipitator before the gases reach the phosphorus condenser.

It should be noted that during the industrial production of phosphorus, it is possible that particles of elemental phosphorus may be emitted. Free phosphorus can exist in air as a vapor because there are upper and lower limits on the ratios in which oxygen and phosphorus react. Consequently, it cannot be assumed that all of the free phosphorus reacts to form phosphorus oxides and suboxides.[27]

3.3 Product Sources

3.3.1 Phosphoric Acids

The types of phosphoric acids produced are ortho- and superphosphoric acids. In the production of orthophosphoric acid, the primary pollutant is phosphoric acid mist. Emission of P_2O_5 is in the range of 0.3 to 5.0 pounds per thousand pounds of elemental phosphorus (P_4) burned.[2] Superphosphoric acid (containing more than 70 percent P_2O_5) is made by dehydrating wet-process acid or by burning elemental phosphorus with air as in the furnace process, except that in the hydrator, the cooling of the combustion gas is done with water or diluted acid sprays. About half of the P_2O_5 drops out of the vessel as superphosphoric acid, while the rest leaves as acid mist at 200° F and is collected as less concentrated acid in an electrostatic precipitator. The diluted acid is sprayed back into the hydrator, and the effluent gas from the precipitator is vented to the stack. Superphosphoric acid is also made from the normal 50 to 55 percent acid. The primary pollutant is the phosphoric acid mist that is not removed by the mist eliminator. The quantity of acid mist can be assumed to be about equal to or less than that vented in the furnace process and is related to the efficiency of the precipitator. Producing phosphoric acid from the wet-process acid is carried out in a high vacuum, which tends to limit emissions. More information can be found in reference 21. In 1967 there were

about 44 plants using the wet process and new plants are being built.[2,68,71]

Flow diagrams of processes used for the manufacture of superphosphoric, wet-process phosphoric, and furnace phosphoric acids are shown in Figures 3, 4, and 5 in the Appendix.

Phosphoric acid ranked second in production value (after ammonia) among the top 73 inorganic chemicals produced in 1965 and ninth in terms of production amount.[9] Table 8 in the Appendix shows the growth of the phosphoric acid industry in the United States.[2]

The location of thermal process phosphoric acid plants in the United States is given in Table 9 in the Appendix.

Phosphoric acids are used for the production of fertilizers, animal feed supplements (mono- and dibasic calcium phosphates), and detergents (sodium and potassium polyphosphates).

3.3.2 Phosphate Fertilizers

Phosphate fertilizers are produced from phosphoric acid and phosphate rocks.

The major types of phosphate fertilizers are:

Diammonium phosphate
Triple superphosphate
Normal superphosphate
Monoammonium phosphate
Ammonium phosphate sulfate
Ammonium phosphate nitrate
Ammonium polyphosphates (solid and liquid)
Nitric phosphates
Other mixed fertilizers
Phosphoric acid applied directly to soil

In the above list, the first three fertilizers are the most important in the order shown. Diammonium phosphate production (48 percent P_2O_5) has been replacing or reducing production of triple superphosphate. In 1965 there were about 48 plants producing Diammonium phosphate.[68] Emission points are shown in Figure 6 in the Appendix.

Triple superphosphate is produced in large plants near phosphate rock deposits. In 1965, there were 16 plants in the United States producing 5.3 million tons per year.[68]

Normal (ordinary) superphosphate fertilizers are produced by reacting sulfuric acid with phosphate rock. In 1965 there were over 200 plants involved in this process, but production is declining as more concentrated P_2O_5 products (triple superphosphate) are being produced. The gases released from acidulation of phosphate rock are the major emissions.[68] Emission points are shown in Figure 7 in the Appendix.

3.3.3 Phosphine

Phosphine is an industrial hazard in (1) the manufacture of acetyleine, where calcium phosphide is an impurity in the calcium carbide used in the process, (2) phosphorus extraction, (3) manufacturing of phosphorus sesquisulfide, and (4) industrial operations with phosphorus-bearing ferrosilicon. Phosphine may, under low pH conditions, be evolved from ferrophosphorus in the production of elemental phosphorus

by the electric furnace process. Phosphine is also generated during the acid pickling of steel which contains small amounts of iron phosphide.

3.4 Other Sources

3.4.1 Oil-Fired Boilers

The fuel oil used most in boilers that produce steam at a rate of 1,000 hp/hr (approximately 34,500 lb/hr) or more is designated as Bunker C, residual, high viscosity, heavy, grade 6, or Pacific Standard 400. Elemental analyses of total particulates in the fly ash from the combustion of residual oil showed in one test that phosphorus (as phosphorus pentoxide) amounted to 0.9 percent of the total solids collected in a laboratory electrical precipitator at 230° F.[65] In small emission sources (less than 1,000 hp/hr), the fly ash loadings are higher than for large sources.[65]

The concentration of total phosphorus in the fly ash from residual-oil-fired boilers has not been measured, nor has the variation in phosphorus emission rates with respect to the variation in other operating parameters been determined.

3.4.2 Iron and Steel Industry

Schueneman et al.[64] reported that phosphorus pentoxide accounted for an average of 0.2 percent of the total weight of fume produced during an entire melting cycle in an electric-arc steel furnace. In a 15-ton-arc steel furnace in which dust collection of about 7 lb/ton of melted steel was obtained,

a typical analysis made in 1959 showed that the dust contained 0.4 percent phosphorus (as phosphorus pentoxide).[16]

A typical analysis of the precipitator dust collected during high-purity oxygen injection from an open-hearth steel furnace (U.S. Steel Corporation, Allegheny County, Pa.) showed a phosphorus pentoxide content of 1.18 percent,[63] while in basic oxygen furnaces, chemical analysis showed that phosphorus pentoxide amounted to 0.3 percent of the dust and fume.

In a recent analysis of oxygen-lanced open-hearth furnace fume, phosphorus pentoxide amounted to 0.5 percent.

In a study[5] of the efficiency of mineral wool filters in collecting acid mists from open-hearth furnaces and other steel producing operations, the concentration of phosphoric acid mist was found to range from 45,000 to 110,000 $\mu g/m^3$.

3.4.3 Transportation Sources[34]

Many organophosphorus derivatives have been widely used to reduce corrosion resulting from the combustion of gasolines and aviation fuels. Approved inhibitors for aviation fuels include alkylamiphosphate and 25 percent octyl phosphate esters, and 25 percent octyl phosphate and 50 percent ammonium alkyl phosphates for gasoline. Ammonium alkyl phosphate is also used an an anti-icing agent. Concentrations of these additives range from 5 to 20 pounds per 1,000 barrels (2.3 to 9 grams per barrel) of aviation fuel.

Phosphorus-containing additives (organophosphorus compounds) are also used as modifiers of combustion chamber

deposits resulting from surface ignition in motor gasolines, as well as inhibitors of spark-plug fouling. The list of such phosphorus compounds commercially available for use in gasoline includes tri-isopropyl phosphite, tri-cresyl phosphate, tri-chloropropyl thionophosphate, dimethyl xylyl phosphate, dimethyl phenyl phosphate, trimethyl phosphate, mixed cresyl phenyl phosphate, and alkyl aryl phosphate.

It is possible that part of the phosphorus in fuel additives is emitted into the ambient air as part of the exhaust gases, and an investigation into this matter appears to be desirable.

3.5 Environmental Air Concentrations

The National Air Sampling Network has not been measuring concentrations of phosphorus and its compounds in the ambient air, and very limited data are available.

Measurements of aerosol and vapor phase phosphates in the Los Angeles atmosphere (1954) indicated that concentration values range from less than detectable limits to 4.23 $\mu g/m^3$ (the average being 1.43 $\mu g/m^3$).[49]

Measurements[33] were made during the period February 14 to April 21, 1966 of air samples from Cincinnati and Fairfax (a suburb of Cincinnati). Single cascade impactors operated for 24-hour periods were used to collect particulates for colorimetric analysis, and both organic and inorganic phosphates in the form of orthophosphate were found. The average phosphate concentrations were 0.22 $\mu g/m^3$ in Cincinnati and

0.31 $\mu g/m^3$ in Fairfax; the corresponding median particle diameters were 2.7 and 3.9 μ respectively. The average particle-size distribution curves for phosphates in Cincinnati and Fairfax are shown in Figure 8 in the Appendix. It can be seen that while the median diameter is in the micron range, about 20 percent of the total mass is in the submicron range.

4. ABATEMENT

In the production of phosphoric acid by the thermal process, the major pollutant is phosphoric acid mist. There are several pollution control devices used which are discussed below; one of the most important factors influencing emission rates is maintenance of the collection equipment.

Scrubbers. The use of packed and open tower scrubbers is a simple and low-cost abatement technique. Collection efficiencies vary from 40 to 95 percent for gas velocities of 2 to 7 ft/sec.[70] Improved efficiency has been achieved by installing wire-mesh mist eliminators after the scrubber. Venturi scrubbers are also used and have higher collection efficiency, ranging from 78 to 98 percent for particle sizes of from 0.5 to 1 μ, respectively.

Cyclones. Cyclones have comparatively low collection efficiencies since they are effective only in the particle-size range of 10 μ and above. In some plants they are compelemented with wire-mesh mist eliminators or used in series with venturi scrubbers. In this case a collection efficiency of up to 99.9 percent of the acid mist can be achieved.[70]

Fiber Mist Eliminators. Plants using glass-fiber mist eliminators operating with gas velocities ranging from 0.4 to 13 ft/sec provide collection efficiencies of 96 to 99.9 percent.[70]

High-Energy Wire-Mesh Contactors. High-energy wire-mesh contactors can operate at high vapor velocities of 20 to

30 ft/sec and provide collection efficiencies that exceed 99.9 percent.[70]

Electrostatic Precipitators. Electrostatic precipitators show high collection efficiencies of 98 to 99 percent, but also high maintenance costs. The factors affecting their performance are the rate of gas flow or temperature and the electrical conditions during their operation.

In a study[24] made by the International Minerals and Chemicals Corporation, it was found that the optimum type of equipment (in the farm chemicals industry) for the control of phosphorus pentoxide (P_2O_5) losses in the fumes is a modified single-stage venturi scrubber followed by an impingement basin. The data reported on emissions from seven plants, together with abatement devices used, are shown in Table 3.

TABLE 3

EMISSIONS AND ABATEMENT DEVICES USED IN SEVEN PHOSPHATE FERTILIZER PLANTS[24]

	Average	Range
Grade manufacturing: 5-10-10 to 10-20-10 production rate, tons/hr	21	15-32
Emission rates		
Solids, lb/hr	36	13-138
Fume, lb/hr	110	31-241
Number of Cyclones*	3	2-4

*Scrubbers used in five out of the seven plants in conjunction with cyclones.

5. ECONOMICS

In the production of phosphoric acid by the thermal process, capital cost of wire-mesh mist eliminators (a recent and effective abatement device) is reported to be approximately $\$1.50/ft^3/min$ of stack gas.[11] Except for electrostatic precipitators, maintenance costs of collection equipment are not high. When a continuous emission monitor is used, excessive emissions can be easily detected. Since the pollutant is also the final output (product) of the process, attempts are usually made to minimize losses from the collection system.[2]

In the farm chemicals industry (phosphate fertilizers, insecticides, etc.) investment in pollution abatement equipment during the last few years have been quite large. Most of this equipment is also used to reduce emissions of other pollutants, such as fluorine and ammonia. For example, Agrico Chemical Company has added a large exhaust and scrubber system costing $581,000 to the control equipment at its phosphate complex in Pierce, Fla., and about $400,000 worth of scrubber systems at several of its phosphate fertilizer plants in other areas. Other companies have been spending comparable amounts for installation of abatement equipment.[24]

There have been large investments in the field of research and development as well as in monitoring systems. According to statistics by the Florida Phosphate Council, the phosphate companies operating in the Polk-East Hillsborough area have spent $48 million to date in research, development,

and installation of air pollution control systems for all air pollutants. Cost of operation and maintenance has been estimated at more than $6 million annually. Cost of installed air pollution equipment varies from 20¢ to $3.50 per cubic foot per minute (cfm).

A study[24] made by the International Minerals and Chemicals Corporation showed that phosphorus pentoxide (P_2O_5) losses varied from 3 to 53 lb/hr. The value of these losses —including those of nitrogen and potassium--varies from $3.63 to $10.66/hr.

It has been estimated[55] that by 1980, the U.S. electric utility industry alone will produce some 45 million tons of fly ash per year, containing 400,000 tons of phosphorus pentoxide. Utilized fly ash represents only 6 percent of the current annual production, while the rest is dumped. Disposal for this type of waste costs about $2 per ton and creates a nuisance as well.

6. METHODS OF ANALYSIS

6.1 Sampling Methods

6.1.1 Free Phosphorus in Air

One of the problems in phosphorus determination is separating free phosphorus from the many phosphorus compounds found in the urban air. A recently developed sampling method draws air through a macro impinger containing 100 ml of xylene at a rate of 1 cfm for 15 minutes. A filter paper attached to the exit catches any fumes which pass through the xylene.[60]

6.1.2 Phosphoric Acid (Plants, Stack Gases)

Sampling of phosphoric acid mist in gas effluents from stacks of phosphoric acid plants (thermal process) can be done by a method based on that used by the U.S. Public Health Service for particulate sampling. The sampling train consists of a probe, cyclone, filter, four impingers, pump, dry-gas meter, calibrated orifice, and manometer, in the order listed.[2] The cyclone is designed so that particulate samples can be separated into two fractions, one having particle diameters less and the other having particles greater than 5 μ. The first two impingers contain water, the third one is dry, and the fourth contains silica gel. When phosphoric acid mist is sampled, the fritted-glass and paper filters are removed, so that all particles passing through the cyclone are collected in the impingers.

6.1.3 Phosphine

In a spectrographic method developed recently for the rapid determination of phosphine, air samples are collected at the rate of 0.5 liter per minute (for about 10 minutes) in a fritted-glass bubbler. The bubbler has a collection efficiency of 86.2 percent at 0.5 liter per minute (75 percent at 1 liter per minute) and contains silver diethyldiocarbamate.[13]

6.2 Quantitative Methods

6.2.1 Phosphorus and Phosphoric Acid

Phosphorus is usually determined and estimated as phosphoric acid expressed as phosphorus pentoxide (P_2O_5). This determination may be done gravimetrically by forming a magnesium pyrophosphate ($Mg_2P_2O_7$) precipitate, titrimetrically, or colorimetrically.[27]

A number of colorimetric methods[32] are available, most of which are based on the development of a blue color when molybdate is treated with a reducing agent. The ring-oven colorimetric method[66] uses as a reagent orthodianisidine molybdate dissolved in glacial acetic acid which contains sodium molybdate. The limit of identification is 0.002 μg and the range 0.05 to 2.0 μg.

The interference of phosphate in fluorimetric procedures became the basis of various studies aimed at developing a highly sensitive method of determining trace amounts of phosphate.[32] One such method, based upon fluorescence quenching

by phosphate of aluminium-morin chelate, permits the determination of 0.5 to 10 µg of phosphate, or 0.5 to 0.1 µg/ml in solution. Ions of a number of elements, especially metals, were found to interfere; and for the method to be specific, phosphate is separated from such ions.[32]

Recently, a method has been developed for the determination of free phosphorus and phosphorus vapor in air that depends on trapping the element and its compounds in xylene. Separation of phosphorus compounds from free phosphorus is done by dissolving the compounds in water and separating the phases, converting the free phosphorus to silver phosphide, oxidizing the phosphide to phosphate, and estimating the phosphate by the molybdenum blue method. Standards are prepared in the range of 0 to 35 µg of phosphorus, and the colorimetric portion of the procedure is carried out with each standard.[60]

Determination of phosphorus in phosphoric acid emitted from stacks of phosphoric acid plants can be made with the ammonium phosphomolybdate colorimetric method, which is based on the spectrophotometric determination of the yellow ammonium phosphomolybdovanadate complex formed when orthophosphate reacts with the reagent in an acid medium. The method is applicable to materials in which phosphorus compounds can be quantitatively oxidized to the orthophosphate form.[22,59,73] Interference comes from (1) certain ions which reduce the color to molybdenum blue, (2) oxalates, tatrates,

and citrates which tend to bleach the color, (3) high concentrations of iron and (4) of dichromate, resulting from the close resemblance of the color of the dichromate ion to the yellow complex ammonium phosphomolybdovanadate. This method is applicable to the determination of total phosphates in the concentration range of about 50 µg to 2,000 µg, with a replication precision of ± 1.0 percent.[70] Another method used for the determination of phosphoric acid in stack gas samples is acid-base titration.

6.2.2 Phosphine

Phosphine in air can be determined by colorimetric methods. Recently a spectrographic method[13] has been developed for the rapid determination of phosphine in the range of 0.1 to 1 ppm. The method uses silver diethyldiocarbamate as reagent and depends on the formation of a complex of phosphine and the reagent with an absorption maximum at 465 mµ. It is necessary to calibrate the procedure by a second method analyzing phosphine as phosphate, since no primary phosphine standards are presently available.

6.2.3 Organophosphorus Pesticides

For the detection of organophosphorus pesticides, gas chromatography has been used in conjunction with a flame-photometric,[39] thermionic,[25] or electron-capture detector. See companion report on pesticides for more details on analytical methods of organophosphorus pesticides.

7. SUMMARY AND CONCLUSIONS

Some forms of phosphorus are toxic to humans and animals. Yellow phosphorus is a protoplasmic poison, while red phosphorus is comparitively nontoxic. Some of the phosphorus inorganic compounds, such as phosphine gas, are highly toxic at concentrations of 400 to 600 ppm, while others—such as tricalcium phosphate—are only mildly so. Many of the organic compounds of phosphorus are extremely toxic. However, very few of the known phosphorus compounds have been investigated with respect to their toxic effects on humans, animals, and plants.

The effect of some phosphorus compounds is the inhibition of cholinesterase, a process which is slow and sometimes cumulative and irreversible. The nervous system is affected by this type of adverse action. Nevertheless, the potential hazard represented by the ambient air concentrations of phosphorus and its compounds is unknown.

The source of almost all phosphorus and phosphorus products is phosphate rock. The major uses of phosphorus compounds are the manufacturing of fertilizers, pesticides, and industrial chemicals. These processes are the major sources of phosphorus emissions in the ambient air. Boilers and furnaces burning some crude oils and coals which contain phosphorus represent secondary emission sources of phosphorus compounds. The production of phosphorus and its primary products

has been increasing annually. Production of phosphate rock in 1966 was more than three times the 1950 level (approximately 100 million long tons), while the production of phosphoric acid shows the highest rate of increase among major industrial acids.

No national data exist on the ambient air concentrations of phosphorus. The few local data available from special studies indicate an average ambient air concentration, in 1954, of 1.43 $\mu g/m^3$.

Abatement of phosphorus emissions includes the use of scrubbers, fiber mist eliminators, high-energy wire-mesh contactors, and electrostatic precipitators.

No information has been found on economic costs of phosphorus air pollution. Investment in air pollution control equipment within the phosphate industry has been relatively high, especially in Florida, where the phosphate fertilizer industry is concentrated. Operating costs of control equipment in this section of the fertilizer industry have been estimated at $6 million annually; this also includes the control of fluoride emissions.

The quantitative determination of phosphorus and its compounds in the environmental air is made by colorimetric and spectrographic mathods. The most frequently used is the molybdenum blue method (colorimetric). Specific methods have been or are being developed for the determination of elemental phosphorus, phosphoric acid, phosphine, and organophosphorus

pesticides. Maximum sensitivities are in the microgram level or below.

Based on the material presented in this report, further studies are suggested in the following areas:

(1) Determination of the long-term exposure effects on man, animals, and plants of phosphorus and its inorganic and organic compounds.

(2) Determination of the concentration of phosphorus in the ambient air, by type of compounds wherever feasible, in the stations monitored by the National Air Sampling Network.

(3) Evaluation of the pollution contribution by phosphorus from selected stationary emission sources, particularly significant local sources, such as the fertilizer industry.

(4) Evaluation of the pollution contribution by phosphorus compounds emitted from transportation sources.

REFERENCES

1. Aldridge, W. N., and J. M. Barnes, Some Problems in Assessing the Toxicity of the "Organophosphorus" Insecticides Towards Mammals, Nature 169:345 (1952).

2. Atmospheric Emissions from Thermal-Process Phosphoric Acid Manufacture, U.S. Dept. of Health, Education, and Welfare, Public Health Service, U.S. Govt. Printing Office, Washington, D.C. (Oct. 1968).

3. Bamberger, E. S., et al., Effect of Phosphorylated Compounds and Inhibitors on CO_2 Fixation by Intact Spinach Chloroplasts, Plant Physiol. 40(5):919 (1965).

4. Bear, F. E., Trace Elements--Progress Report on Research, Agri. and Food Chem. 2:244 (1954).

5. Billings, C. E., et al., Simultaneous Removal of Acid Gases, Mists, and Fumes with Mineral Wool Filters, J. Air Pollution Control Assoc. 8(3):185 (1958).

6. Bingham, F. T., et al., Effect of Phosphorus Fertilization of California Soils on Minor Element Nutrition of Citrus, Soil Sci. 86:24 (1958).

7. Bingham, F. T., et al., Solubility and Availability of Micronutrients in Relation to Phosphorus Fertilization, Soil Sci. Soc. Amer. Proc. 24:209 (1960).

8. Brewer, R. F., et al., Influence of N-P-K Fertilization on Incidence and Severity of Oxidant Injury to Mangels and Spinach, Soil Sci. 92(5):298 (1961).

9. Chemical Origins and Markets, Stanford Research Institute, Menlo Park, Calif. (1967).

10. Chester, C. G. C., et al., The Role of Zinc in Plant Metabolism, Biol. Rev. 26:239 (1951).

11. Collector 99.9% Efficient: Pressure Drop Moderate, Chem. Processing (Chicago), p. 48 (1966).

12. Dathe, R. A., et al., Electrocardiographic Changes Resulting from Phosphorus Poisoning, Am. Heart J. 31:98 (1946).

13. Dechant, R., et al., Determination of Phosphine in Air, Am. Ind. Hyg. Assoc. J. 27:75 (1966).

14. Diaz-Rivera, R. S., et al., Acute Phosphorus Poisoning in Man: A Study of 50 Cases, Medicine 29:269 (1950).

15. Diaz-Rivera, R. S., et al., The Electrocardiographic Changes in Acute Phosphorus Poisoning in Man, Am. J. Med. Sci. 241:758 (1961).

16. Douglas, I. H., Direct Fume Extraction and Collection Applied to a Fifteen Ton Arc Furnace, Iron and Steel Institute, Special Report No. 83, pp. 144-149 (Sept. 1963).

17. DuBois, K. P., et al., Influence of Induction of Hepatic Microsomal Enzymes by Phenobarbital on Toxicity of Organic Phosphate Insecticides, Proc. Soc. Exptl. Biol. Med. 129: 699 (1968).

18. Einbrodt, H. J., et al., Die chemische Zusammensetzung der in den Lungen und regionaren Lymphknoten abgelagerten Staube bei Grubenpferden, Beitr. Silikose-Forsch. 91:29 (1967).

19. Fedor, W. S., Inorganics Hold Rapid Pace, Chem. Eng. News pp. 80A-91A (Sept. 4, 1967).

20. Fedor, W. S., Inorganics Steady in a Slowdown, Chem. Eng. News pp. 98A-110A (Sept. 2, 1968).

21. Gandevia, B., et al., Relevance of Respiratory Symptoms and Signs to Ventilatory Capacity Changes After Exposure to Grain Dust and Phosphate Rock Dust, Brit. J. Ind. Med. 23:181 (1966).

22. Guinland, K. P., et al., Spectrophotometric Determination of Phosphorus as Molybdovanadophosphoric Acid, Anal. Chem. 27:1626 (1955).

23. Henderson, Y., et al., Noxious Gases and the Principles of Perspiration (New York: Reinhold, 1943).

24. Industry Answers the Challenge, Farm Chem. pp. 21, 24, 26, 28 (June 1967).

25. Ives, N. F., et al., Pesticide Residues, Investigation of Thermionic Detector Response for the Gas Chromatography of P, N, As, and Cl Organic Compounds, J. Assoc. Off. Analyt. Chem. 50(1):1 (1967).

26. Jacobs, M. B., The Analytical Chemistry of Industrial Poisons, Hazards, and Solvents (New York: Interscience, 1949).

27. Jacobs, M. B., The Analytical Toxicology of Industrial Inorganic Poisons (New York: Interscience, 1967).

28. Jacobson, K. H., The Acute Toxicities of Some Intermediates in GB Manufacture, Report No. 17, Army Chemical Center, Md. (1953).

29. Kloos, E. J., Gas Masks for Respiratory Protection Against Phosphine, U.S. Government Printing Office (1966).

30. Kopcznski, W., et al., Mental and Sexual Disorders as a Sequela of Tricresyl Orthophosphate Poisoning, Psychiatria Polska 1(3):267 (1967).

31. Kosalapoff, G. M., Organosphosphorus Compounds (New York: Wiley, 1950).

32. Land, D. B., et al., A Fluoremetric Method for Determining Trace Quantities of Phosphate, Mikrochim. Acta (Vienna) 6:1013 (1966).

33. Lee, R. E., et al., Size Determination of Atmospheric Phosphate, Nitrate, Chloride, and Ammonium Particulate in Several Urban Areas, U.S. Dept. of Health, Education, and Welfare, Public Health Service, Cincinnati, Ohio (1968).

34. Lodwick, J. R., Chemical Additives in Petroleum Fuels: Some Uses and Action Mechanisms, Inst. Petrol. J. 50(491): 297 (1964).

35. Loneragen, J. F., The Effect of Applied Phosphate on the Uptake of Zinc by Flax, Australian J. Sci. Res. Ser. B. 4: 108 (1951).

36. Lovelace, C. J., et al., In Vitro Effects of Fluoride on Tricarboxylic Acid Cycle Dehydrogenases and Oxidative Phosphorylation: Part I., J. Histochem. Cyctochem. 15(4):195 (1967).

37. Mackworth, et al., Biochem. J. 42:91 (1948).

38. The Merck Index, 8th ed. (Rahway, N.J.: Merck, 1968).

39. Midwest Research Institute for Division of Pesticides, U.S. Food and Drug Administration (Report to be published).

40. Millikan, C. R., Effects of Phosphates on the Development of Zinc Deficiency Symptoms in Flax, J. Dept. Agr. Victoria 45:273 (1947).

41. Minerals Yearbook, Bureau of Mines, U.S. Govt. Printing Office, Washington, D.C. (1950).

42. Minerals Yearbook, Bureau of Mines, U.S. Govt. Printing Office, Washington, D.C. (1955).

43. Minerals Yearbook, Bureau of Mines, U.S. Govt. Printing Office, Washington, D.C. (1960).

44. Minerals Yearbook, Bureau of Mines, U.S. Govt. Printing Office, Washington, D.C. (1965).

45. Minerals Yearbook, Bureau of Mines, U.S. Govt. Printing Office, Washington, D.C. (1966).

46. Minerals Yearbook, Bureau of Mines, U.S. Govt. Printing Office, Washington, D.C. (1967).

47. Moeschlin, S., Poisoning, Diagnosis, and Treatment (New York: Grune & Stratton, 1965).

48. Nachmansohn, D., Chemical Factors Affecting the Physiology and Pathology of Nervous Function, Contract DA/49/007/MD/740, Armed Forces Technical Information Agency, Arlington, Va. (1959).

49. Neiburger, M., "Meteorological Aspects of Oxidation Type Air Pollution," in The Rossby Memorial Volume (New York: The Rockefeller Institute Press in assoc. with Oxford Univ. Press, p. 158, 1959).

50. Newburger, R. A., et al., Phosphorus Poisoning with Recovery Accompanied by Electrocardiographic Changes, Am. J. Med., 4:927 (1948).

51. Oettengen, W. F. von, Poisoning: A Guide to Clinical Diagnosis and Treatment (Philadelphia: Saunders, 1958).

52. Patty, F. A. (Ed.), Industrial Hygiene and Toxicology, vol. II (New York: Interscience, 1949).

53. Pietras, R. J., et al., Phosphorus Poisoning Simulating Acute Myocardial Infarction, Arch. Internal Med. 122:430 (1968).

54. Properties and Essential Information for Safe Handling and Use of Phosphoric Acid, Chemical Safety Data Sheet SD-70, Manufacturing Chemists Association, Washington, D.C. (1958).

55. Pursglove, J., Jr., Fly Ash in 1980, Coal Age, pp. 84-85 (Aug. 1957).

56. Reimann, H. A., et al., Tricalcium Phosphate Crystallosis, J. Am. Med. Assoc. 189:195 (1964).

57. Reimann, H. A., et al., Experimental Tricalcium Phosphate Crystallosis, Arch. Environ. Health 10:33 (1965).

58. Rogers, L. H., et al., Zinc Uptake by Oats as Influenced by Applications of Lime and Phosphate, J. Am. Soc. Agron. 40: 563 (1948).

59. Rogers, R. N., Determination of Phosphate by Differential Spectrophotometry, Anal. Chem. 32:1050 (1960).

60. Rushing, D. E., A Tentative Method for the Determination of Elemental Phosphorus in Air, U.S. Dept. of Health, Education, and Welfare, Public Health Service, Salt Lake City, Utah (1968).

61. Ryazanov, V. A. (Ed.), Limits of Allowable Concentrations of Air Pollutants, Translated from the Russian by B. S. Levine, Book 2 (1955), Clearinghouse for Federal Scientific and Technical Information, Springfield, Va.

62. Sassi, C., Occupational Poisoning due to Phosphorus Trichloride, J. AMA Ind. Hyg. & Occ. Med. 7:178 (1953).

63. Schneider, R. L., Engineering, Operation and Maintenance of Electrostatic Precipitators on Open Hearth Furnaces, J. Air Pollution Control Assoc. 13(8):348 (1963).

64. Schueneman, J. J., et al., Air Pollution Aspects of the Iron and Steel Industry, U.S. Dept. of Health, Education, and Welfare, Public Health Service, Cincinnati, Ohio (June 1963).

65. Smith, W. S., Atmospheric Emissions from Fuel Oil Consumption, U.S. Dept. of Health, Education, and Welfare, Public Health Service, Cincinnati, Ohio (Nov. 1962).

66. Stern, A. C. (Ed.), Air Pollution, vol I (New York: Academic Press, 1968).

67. Stern, A. C. (Ed.), Air Pollution, vol. II (New York: Academic Press, 1968).

68. Stern, A. C. (Ed.), Air Pollution, vol. III (New York: Academic Press, 1968).

69. Stout, P. R., et al., Molybdenum Nutrition of Crop Plants, Plant Soil 3:51 (1951).

70. Striplin, M. M., Development of Processes and Equipment for Production of Phosphoric Acid, Chemical Engineering Report No. 2, Tennessee Valley Authority (1948).

71. Superphosphate: Its History, Chemistry, and Manufacture, U.S. Dept. of Agriculture, U.S. Govt. Printing Office, Washington, D.C. (Dec. 1964).

72. Tabershaw, I. R., et al., Sequelae of Acute Organic Phosphate poisoning, J. Occupational Med. 8(1):5 (1966).

73. Talvitic, N. A., et al., Spectrophotometric Determination of Phosphorus as Molybdovanadophosphorus Acid, Application to Airborne Particulate Matter, Anal. Chem. 34:866 (1962).

74. Thaker, E. J., et al., Occurrence of Mineral Deficiencies and Toxicities, Soil Sci. 85(ii):87 (1958).

75. Thomson, L. M., Soil and Soil Fertility (New York: McGraw-Hill, 1952).

76. Threshold Limit Values for 1967, Adopted at the 29th Annual Meeting of the American Conference of Governmental Industrial Governmental Hygienists, Chicago, Ill. (May 1-2, 1967).

77. Trakhtenberg, I. M., The Toxicity of Vapors of Organic Mercury Compounds (Ethylmercuric Phosphate and Ethylmercuric Chloride) in Acute and Chronic Intoxication, Survey of U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 3:205 (1960).

78. Waters, R. F., et al., Extraction of Germanium and Gallium from Coal Fly Ash and Phosphorus Furnace Flue Dust, Bureau of Mines, Report No. 6940 (Apr. 1967).

79. Weeks, M. H., et al., Acute Vapor Toxicity of Phosphorus Oxychloride, Phosphorus Trichloride, and Methyl Phosphonic Dichloride, Am. Ind. Hyg. Assoc. J. 25(5):470 (1964).

OTHER REFERENCES

Coykendall, J. W., et al., New High-Efficiency Mist Collector, J. Air Pollution Control Assoc. 18(5):315 (1968).

Diem, K. (Ed.), Documenta Geigy, Scientific Tables, 6th ed. (Adsley, N.Y.: Geigy Chemical Corp., 1962),

Jewell, J. P., Control of Fluoride Emissions, Proceedings of 5th Annual Sanitary and Water Resources Engineering Conference, Nashville, Tenn., p. 226 (June 1965).

Mayer, M., A Compilation of Air Pollutant Emission Factors for Combustion Processes, Gasoline Evaporation, and Selected Industrial Processes, Technical Assistance Branch, Division of Air Pollution, U.S. Dept. of Health, Education, and Welfare, Public Health Service, Cincinnati, Ohio (May 1965).

Schutte, K. H., The Biology of Trace Elements (Philadelphia: Lippincott, 1964).

Senchuk, V. S., The Effect of Atmospheric Ultraviolet Radiation Insufficiency on Mineral Metabolism, USSR Literature on Air Pollution and Related Occupational Diseases 1:111 (1960).

Teller, A. J., Control of Gaseous Fluoride Emissions, Chem. Eng. Progr. 63(3):75 (1967).

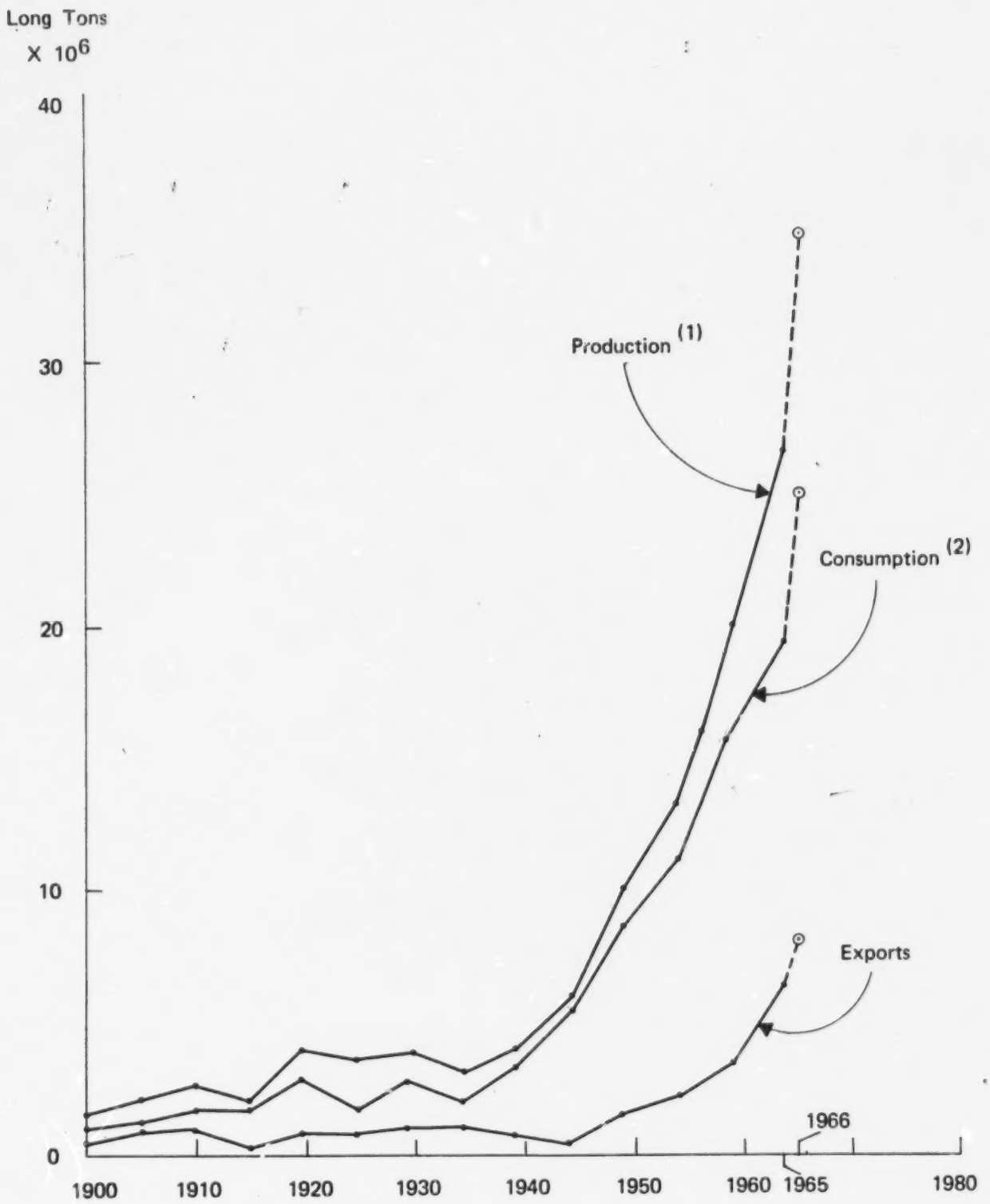
Weinstein, L. H., et al., Automated Analysis of Phosphorus-Containing Compounds in Biological Materials. I. A Quantitative Procedure, Contrib. Boyce Thompson Inst. 22(7):389 (1964).

Weinstein, L. H., et al., Automated Analysis of Phosphorus-Containing Compounds in Biological Materials. II. Acid-Soluble Nucleotides, Short Communications, Anal. Biochem. 2 (1):155 (1965).

Wett, T. W. (Ed.), Competitors Collaborate to Choke Off Air Pollution, Chem. Processing 31(1):27 (1968).

Woltz, S. S., Distinctive Effects of Root Versus Leaf Acquired Fluorides, Proc. of Florida State Horticultural Society (Miami, Fla.) 77:516 (1964).

APPENDIX



(1) Marketed production (sold or used)
(2) Apparent consumption (=production + imports-exports)

FIGURE 1

Marketed Production, Apparent Consumption, and Exports of Phosphate Rock, 1900-66[41-45,71]

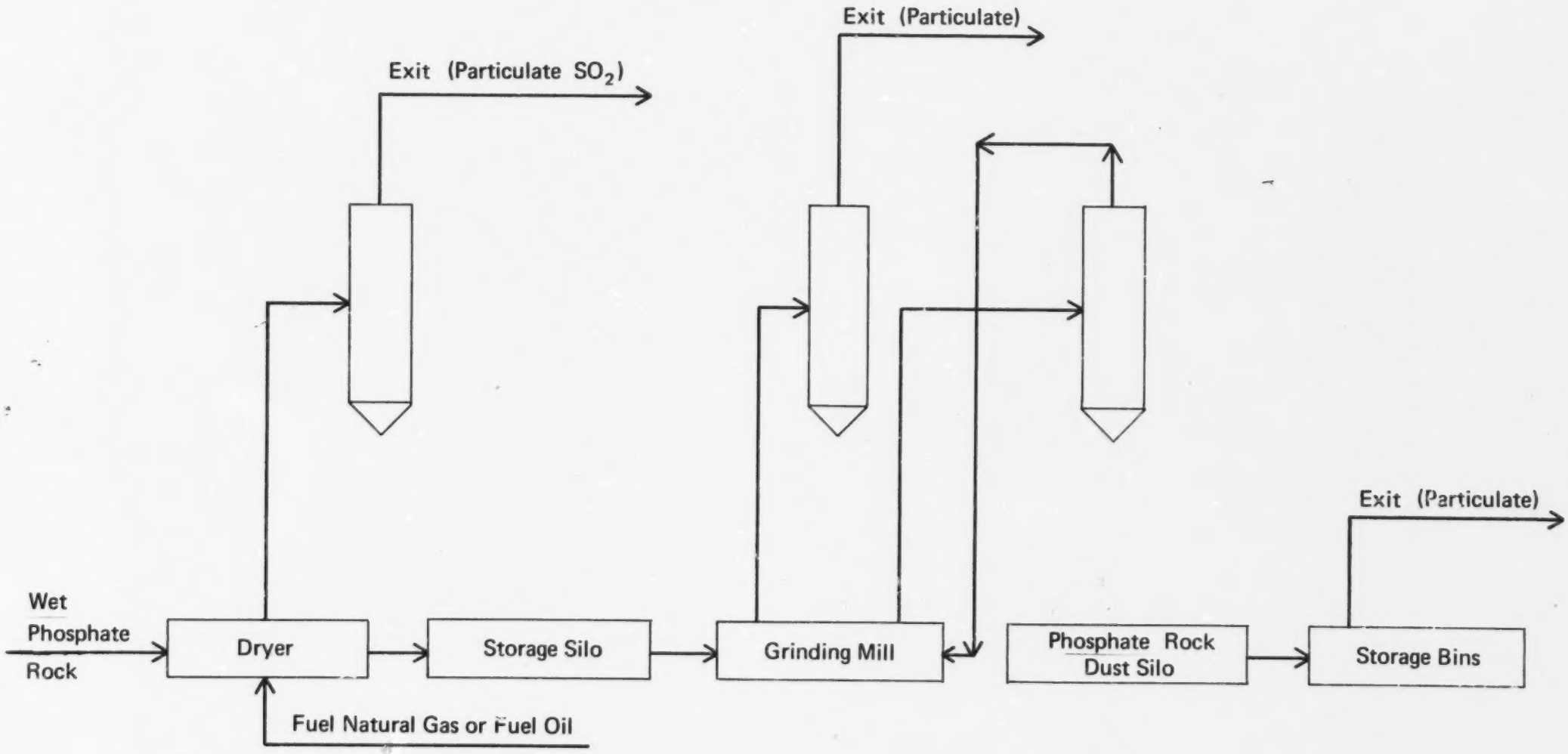


FIGURE 2

Flow Diagram of Phosphate Rock Storage and Grinding Facilities[68]

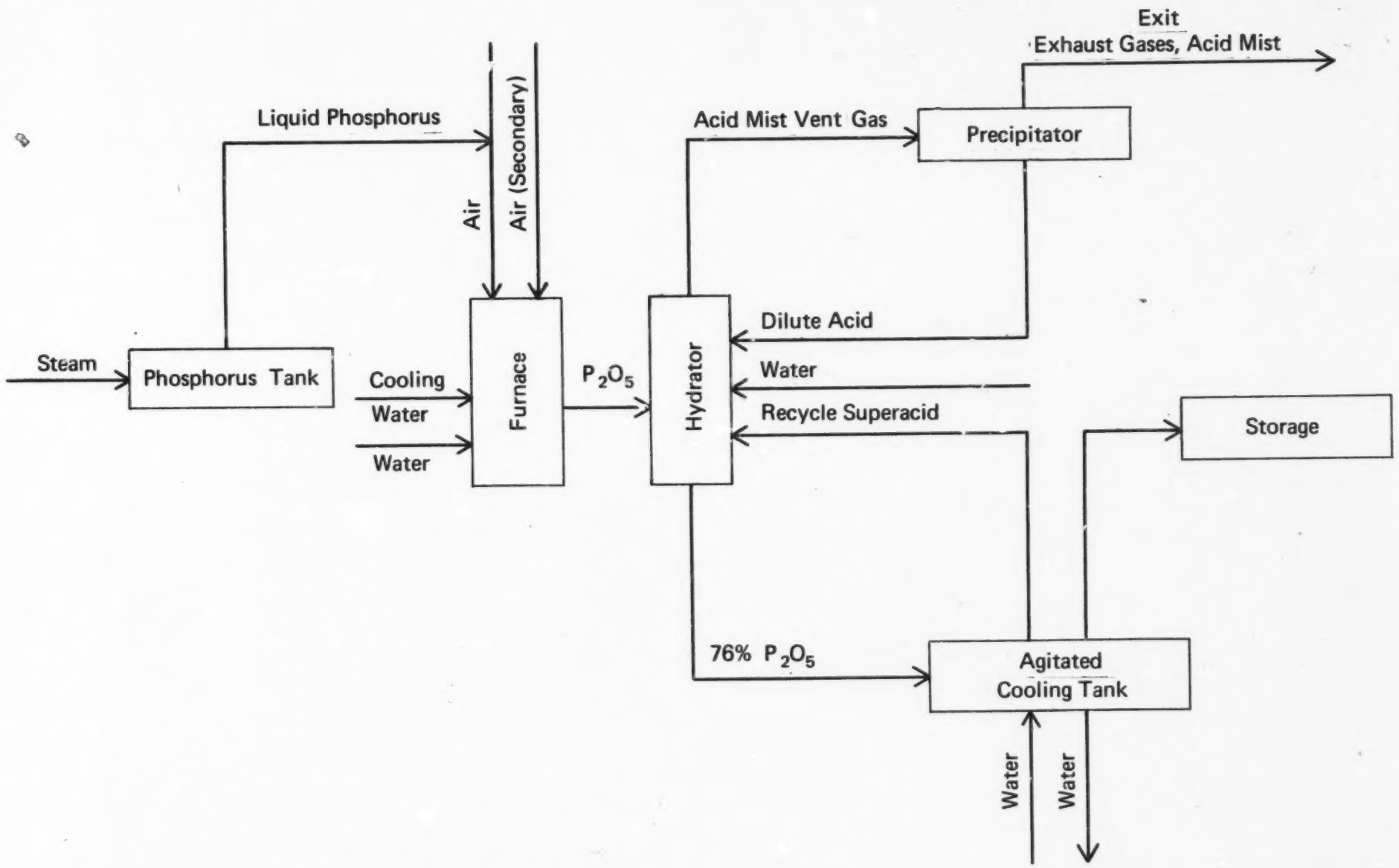


FIGURE 3

Flow Diagram of Process for Production of Superphosphoric Acid[68]

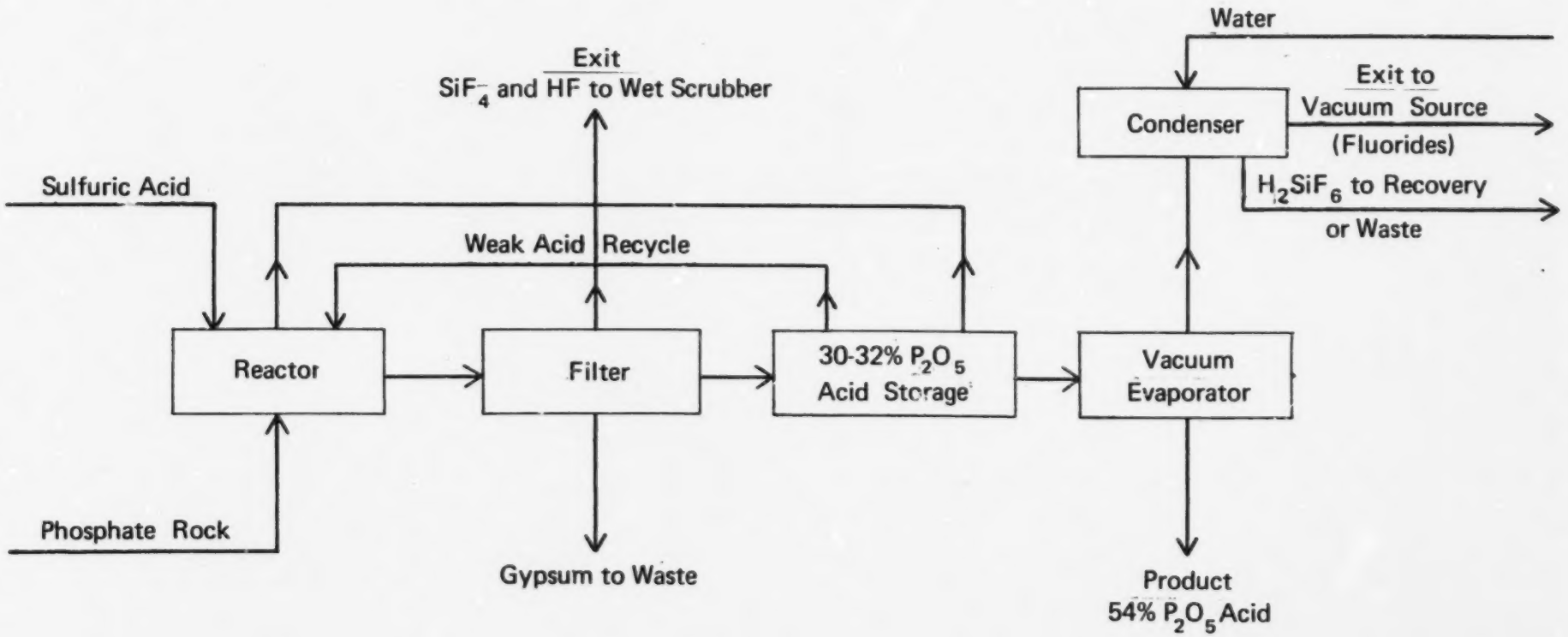


FIGURE 4

Flow Diagram of Wet Process Phosphoric Acid Plant[68]

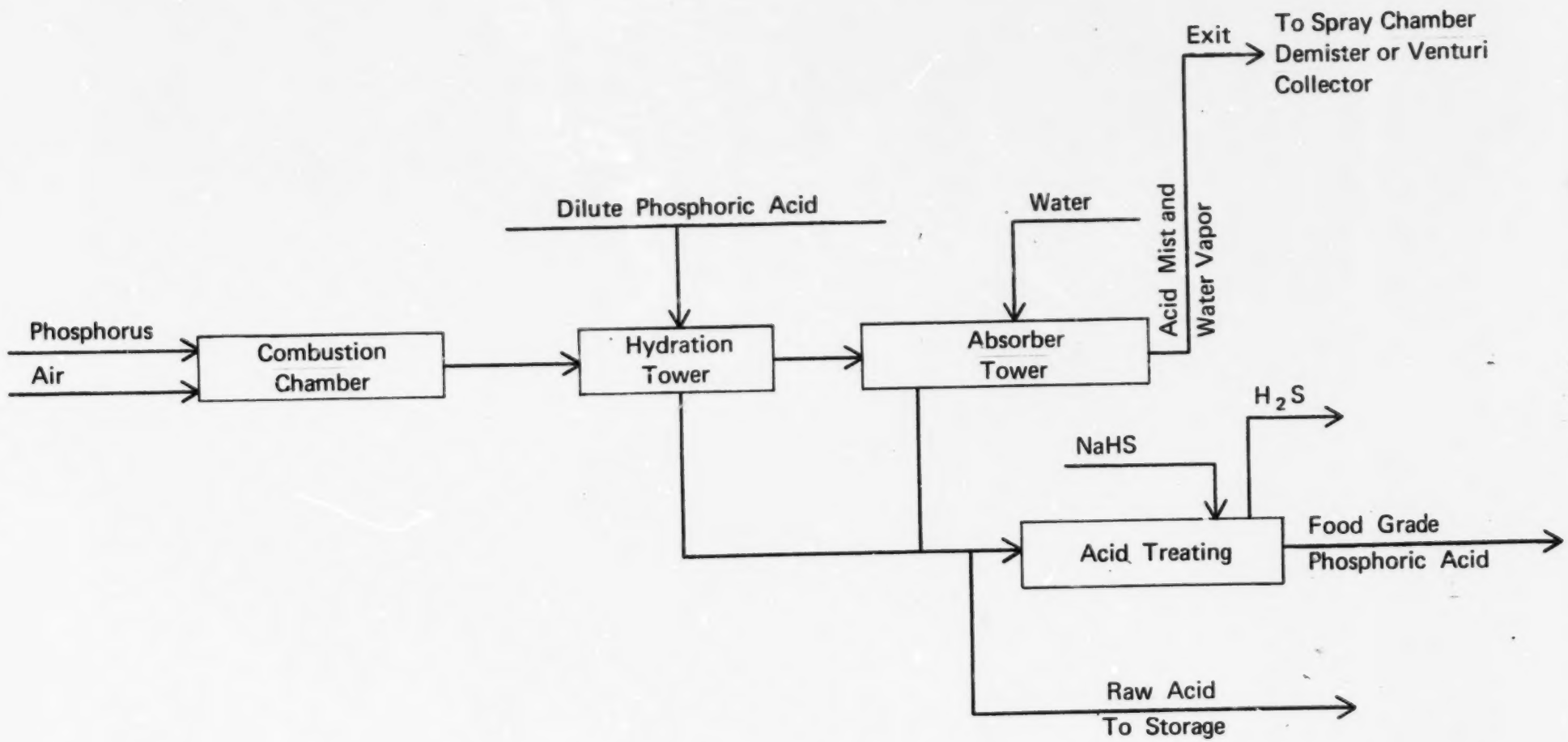


FIGURE 5

Flow Diagram of Furnace Phosphoric Acid Plant[68]

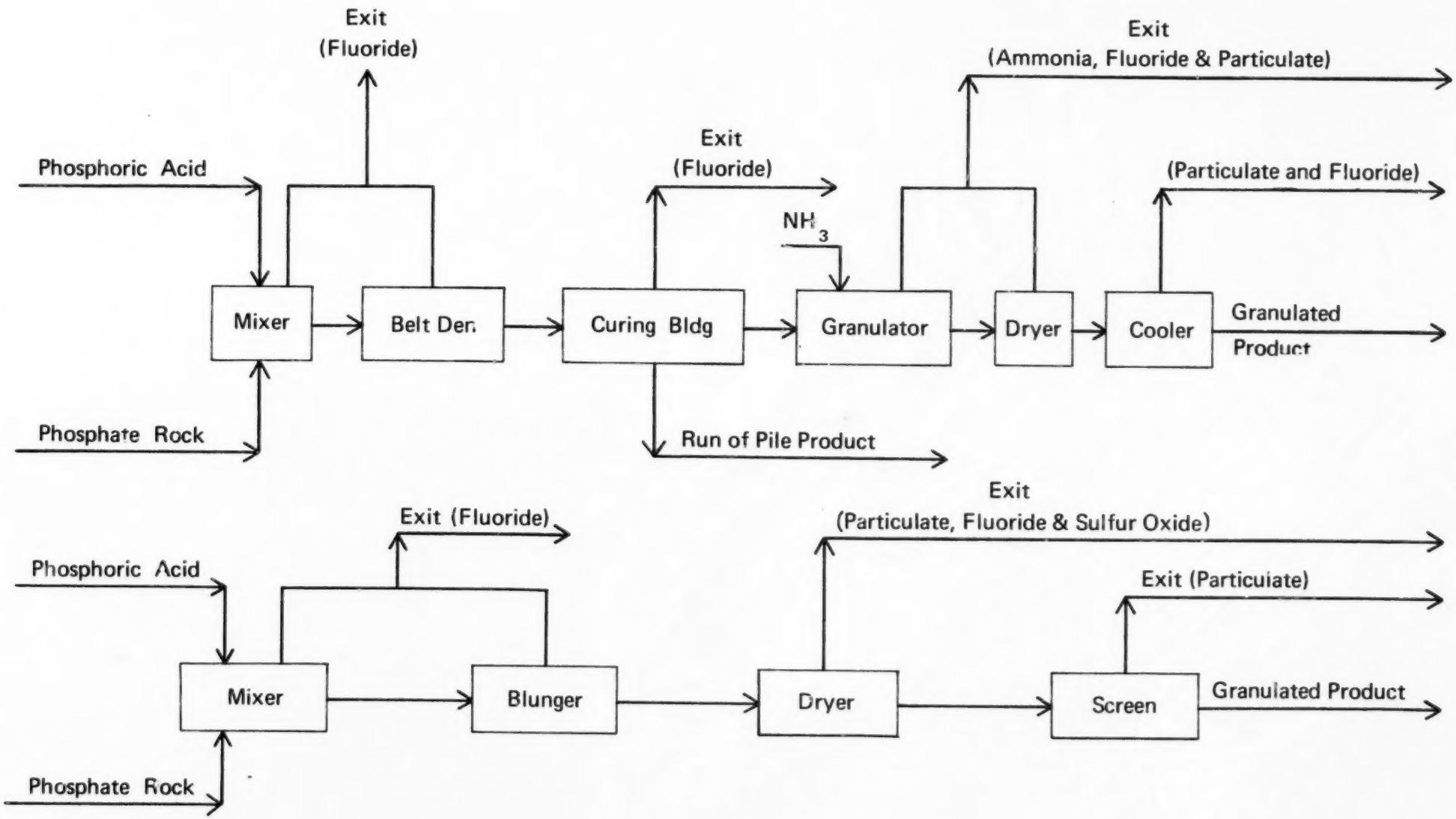


FIGURE 6

Flow Diagram: Production of Run of Pile and Granular Triple Superphosphate[68]

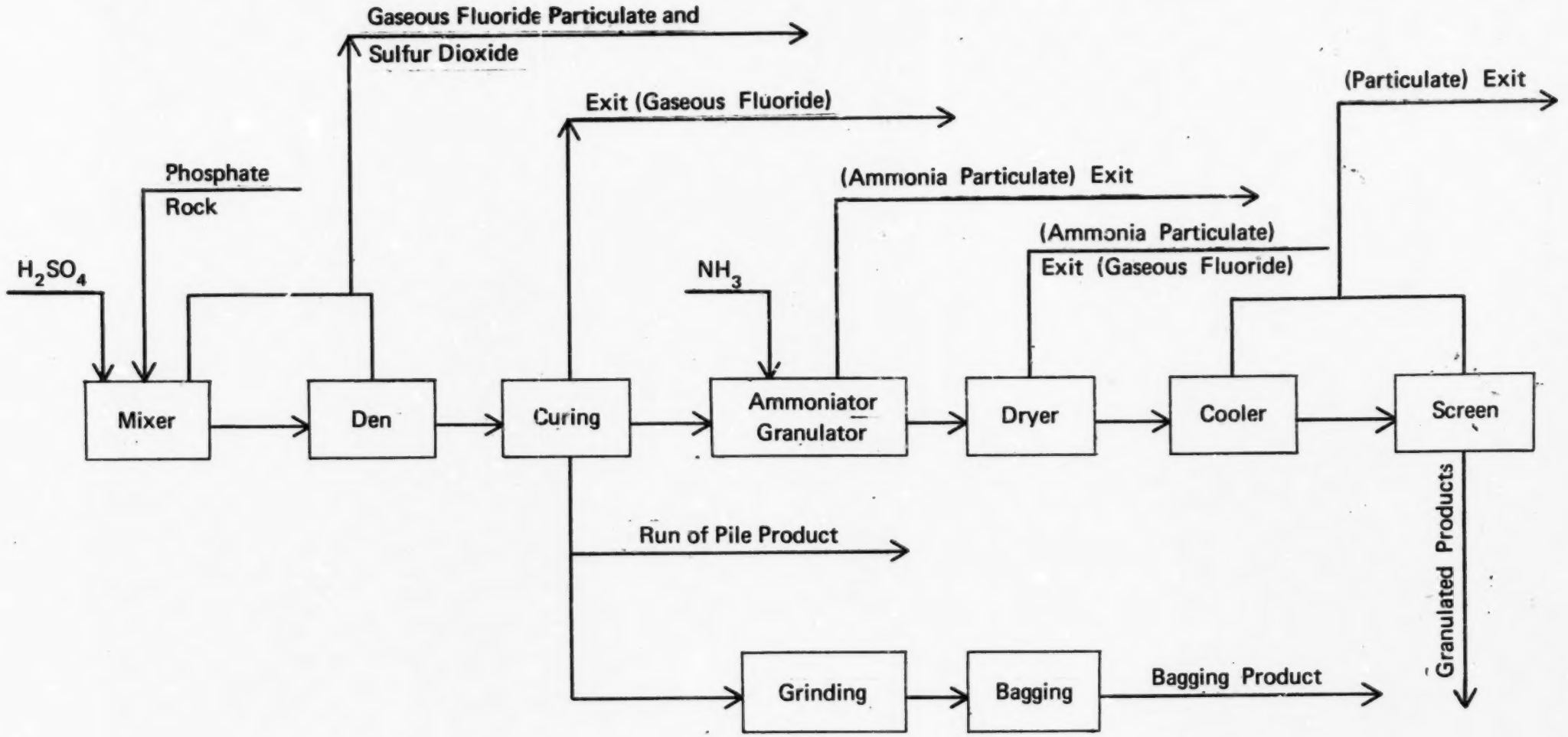


FIGURE 7

Flow Diagram of Normal Superphosphate Plant[68]

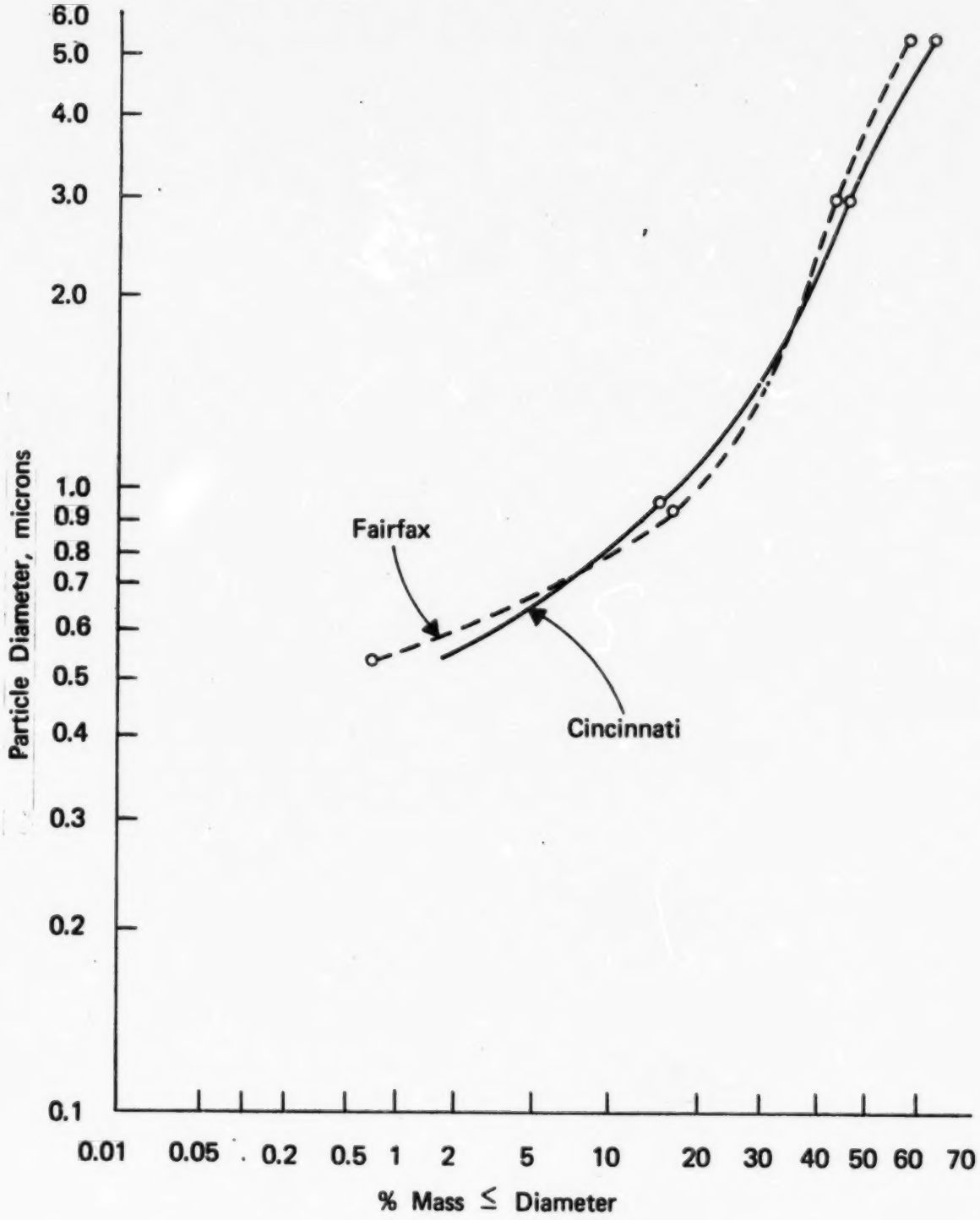


FIGURE 8

Cumulative Particle Size Distribution of Phosphate in Cincinnati and Fairfax, Ohio[33]

APPENDIX

TABLE 4

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS[38]

Compound	Properties	Toxicity	Uses
Phosphorus P	White phosphorus: colorless or yellowish crystalline solid mp 44.1° bp 280° volatile Black phosphorus: crystals Red phosphorus: red to violet powder Sublimes at 416°	White phosphorus: ingestion of even small amounts may produce severe gastrointestinal irritation, bloody diarrhea, liver damage, skin eruptions, cliguria, circulatory collapse, coma, convulsions, death. The approximate fatal dose of white (also called yellow) phosphorus is 50 mg. External contact may cause severe burns. Chronic poisoning (from ingestion or inhalation) produces spontaneous fractures, anemia, weight loss Red phosphorus is relatively nontoxic unless it contains the white form as an impurity	Red phosphorus: in pyrotechnics, manufacture of safety matches, organic synthesis; in manufacture of phosphoric acid, phosphine phosphoric anhydride, phosphorus pentachloride, phosphorus trichloride; in manufacture of fertilizers, pesticides, incendiary shells, smoke bombs, tracer bullets. Med. use: formerly in rickets, other bone diseases, and degenerative disorders of central nervous system
Phosphamidon $C_{10}H_{19}ClNO_5P$	Oil bp 162°	Cholinesterase inhibitor	Systemic insecticide

(continued)

APPENDIX

TABLE 4 (Continued)

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS

Compound	Properties	Toxicity	Uses
Phosphine PH_3	Gas Odor of decaying fish bp -87.7^{o} mp -133^{o}	Inhalation causes pain in region of diaphragm, a feeling of coldness, vertigo, dyspnea, bronchitis, edema, lung damage, convulsions, coma, death. Lethal dose for rats in air, 60 ppm	
Phosphocreatine $C_4H_{10}N_3O_5P$			Med. use: In sodium salt to treat fatigue.
Phosphomolybdic acid $20MoO_3 \cdot 2H_3PO_4 \cdot 48H_2O$	Bright yellow crystals		In weighting silks; as reagent for alkaloids, uric acid, xanthine, creatinine, some metals, with hematoxylin as nerve stain in microscopy
Phosphonium iodide PH_4I	Large transparent colorless crystals	Readily liberates phosphine, which is highly toxic, and hydriodic acid, which is a strong irritant	In laboratory preparation of phosphine

(continued)

TABLE 4 (Continued)

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS

Compound	Properties	Toxicity	Uses
Phosphoric acid H_3PO_4	Unstable orthorhombic crystals mp 42.35°	Concentrated solutions are irritating to the skin and mucous membranes	In the manufacture of superphosphates for fertilizers, other phosphate salts, polyphosphates, detergents; as acid catalyst in making ethylene, purifying hydrogen peroxide; as acidulant and flavor in beverages of the soft drink type; in dental cements; in process engraving; in rustproofing of metals before painting; in coagulating rubber latex; as analytical reagent. Med. use: has been used (dilute form) as gastric and urinary acidifier; as dressing for removal of necrotic debris of burns. Vet. use: diluted phosphoric acid has been used in treating lead poisoning
Phosphoric acid $(HPO_3)_n$	Colorless, transparent, glass-like solid		In dentistry for making zinc oxyphosphate cement; as reagent in chemical analysis
Phosphorous acid H_3PO_3	White crystalline mass, garlic-like taste		

(continued)

TABLE 4 (Continued)

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS

Compound	Properties	Toxicity	Uses
Phosphorus hemitriselenide P_2Se_3	Dark red mass Decomposes by heat		
Phosphorus oxybromide $POBr_3$	Thin plates Faintly orange mp 56° bp 193°		
Phosphorus oxychloride $POCl_3$	Colorless, clear liquid bp 107°	Intensely irritating to eyes, skin, mucous membranes. Inhalation may cause pulmonary edema	As chlorinating agent, especially to replace oxygen in organic compounds; as solvent in cryoscopy
Phosphorus pentabromide PBr_5	Yellow crystalline mass mp above 100°	Toxic and corrosive	
Phosphorus pentachloride PCl_5	White to pale yellow mp 148° bp 160°	Toxic and corrosive	As catalyst in manufacture of acetylcellulose; for replacing hydroxyl groups by Cl, particularly in converting acids into acid chlorides
Phosphorus pentafluoride PF_5	Colorless gas mp -93.8° bp -84.6°	Intensely irritating to skin, eyes, mucous membranes. Inhalation may cause pulmonary edema	

(continued)

TABLE 4 (Continued)

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS

Compound	Properties	Toxicity	Uses
Phosphorus pentaselenide P_2Se_5	Black needles Decomposes in steam and boiling water		
Phosphorus pentasulfide P_2S_5	Light yellow crystalline mass mp 280° bp 523°	On contact with water, decomposes to form hydrogen sulfide. In air, forms phosphorus pentoxide, which is a strong irritant	In manufacturing safety matches, ignition compounds, and for introducing sulfur into organic compounds
Phosphorus pentoxide P_2O_5	Very deliquescent crystals mp 580-585°	Strong irritant, corrosive	As drying and dehydrating agent, condensing agent in organic synthesis
Phosphorus sulfochloride $PSCl_3$	Fuming liquid bp 125°	Strong irritant	
Phosphorus tribromide PBr_3	Colorless liquid bp 175°	Toxic and corrosive	
Phosphorus trichloride PCl_3	Colorless liquid mp -112° bp 76°	Highly irritating and corrosive to skin, mucous membranes	Same uses as phosphorus oxychloride; in manufacturing of $POCl_3$, PCl_5, and in producing iridescent metallic deposits

(continued)

APPENDIX

TABLE 4 (Continued)

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS

Compound	Properties	Toxicity	Uses
Phosphorus trifluoride PF_3	Colorless gas mp -151.5^{o} bp -101.8^{o}	Intensely irritating to skin, eyes, mucous membranes. Inhalation may cause pulmonary edema	
Phosphorus trioxide P_2O_3	Transparent monoclinic crystals mp 23.8^{o} bp 173.1^{o}		
Phosphorus triselenide P_4Se_3	Orange-red crystals mp 242^{o} bp $360-400^{o}$		
Phosphorylcholine $[(CH_3)_3{}^{+}NCH_2-CH_2OPO_3-H_2]Cl$			Med. use: As the magnesium salt in hepatobiliary dysfunction
Phosphotungstic acid $24WO_3 \cdot 2H_3-PO_4 \cdot 48H_2O$	White or slightly yellow-green		As reagent for alkaloids and many other nitrogen bases, phenols, albumin, peptone, aminoacids, uric acids, urea, blood, carbohydrates

(continued)

TABLE 4 (Continued)

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS

Compound	Properties	Toxicity	Uses
5'-Adenylic acid (Phosaden) $C_{10}H_{14}N_5O_7P$	mp 196-200°		Med. use: adenyl compounds have been reported to produce coronary dilation, bradycardia, hypotension, leukocytosis
Phosdrin $C_7H_{13}O_6P$	Yellow liquid mixture bp 106-107.5°	Cholinesterase inhibitor LD_{50} orally in mice: 9.0 mg/kg; in rats 4.0 mg/kg	As systemic insecticide
Alpha-Hydroxybenzylphosphinic acid (Phosilit) $C_7H_9O_3P$	mp 110°		Med. use: to counteract toxic effects of X-ray therapy; the sodium salt as nutritive in convalescence
Tonophosphan $C_9H_{13}NO_2PNa \cdot 3H_2O$	Scales or needles Soluble in cold water		Med. use: As tonic
Diethyl p-nitrophenyl phosphate (phosphacol) $C_{10}H_{14}NO_6P$	Oily liquid		Med. use: As a parasympathomimetic and miotic
Aluminum phosphate $AlPO_4$	White infusible powder mp above 1,460°		As cement in admixture with calcium sulfate and sodium silicate; as flux for ceramics; in dental cements; for special glasses. Used in pharmacy as the gel or dried gel. Med. use: as gastric antacid

(continued)

APPENDIX

TABLE 4 (Continued)

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS

Compound	Properties	Toxicity	Uses
Aluminum phosphide AlP	Dark gray or dark yellow	Must be protected from moist air since it reacts readily to produce phosphine, which is highly toxic	As source of phosphine: in semiconductor research
Sodium phosphate, dibasic Na_2HPO_4	Hygroscopic powder	Anhydrous form may cause mild irritation to skin, mucous membranes; causes purging when taken internally	As a mordant in dyeing; for weighting silk, in tanning; in manufacture of enamels, ceramics, detergents, boiler compounds; as fireproofing agent; in soldering and brazing instead of borax; as reagent and buffer in analytical chemistry. Med. use: As mild saline cathartic; has been used in phosphorus deficiency and in lead poisoning. Vet. use: As laxative for foals, calves
Glycerophosphoric acid $C_3H_9O_6P$	Clear syrupy liquid mp -25°		Absolute acid used to manufacture certain glycerophosphates or to impart taste to solutions of glycerophosphates which are generally used medicinally

(continued)

TABLE 4 (Continued)

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS

Compound	Properties	Toxicity	Uses
Lecithin	Waxy mass Insoluble in water		Is an edible and digestible surfactant and emulsifier of natural origin. Used in margarine, chocolate, and in the food industry in general; in pharmacueticals and cosmetics; and for other industrial uses, such as treating leather and textiles. Med. use: as lipotropic agent
Phosphoric acid 2,2 dichlorovinyl dimethyl ester (DDVP) $C_4H_7Cl_2O_4P$	Nonflammable liquid	A cholinesterase inhibitor LD_{50} in rats 70 mg/kg	As insecticide for the control of agricultural and household pests. Used as 0.5% spray or 0.5% bait. Vet. use: for gastrointentinal worms in swine, ruminants, horses
Phosphoric acid dimethyl ester, ester with cis-3-hydroxy-N,N-dimethylcrotonamide (Bidrin) $C_8H_{16}NO_5P$	Brown liquid	Highly toxic See Parathion A cholinesterase inhibitor LD_{50} orally in rats 22 mg/kg	As insecticide

(continued)

APPENDIX

TABLE 4 (Continued)

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS

Compound	Properties	Toxicity	Uses
Cupric phosphate $Cu_3(PO_4)_2$	Blue or olive orthorhombic crystals		As fungicide; as catalyst for organic reactions; in fertilizer; as emulsifier; as corrosion inhibitor for H_3PO_4; protectant for metal surfaces against oxidation
Parathion O, O-diethyl O-p-nitriphenyl phosphorothionate $C_{10}H_{14}NO_5PS$	Yellowish liquid	Highly toxic Acute: anorexia, nausea, vomiting, diarrhea, excessive salivation, pupillary constriction, bronchoconstriction, muscle twiching, convulsions, coma, respiratory failure. Effects are cumulative. Special precautions necessary to prevent inhalation and skin contamination	As agricultural insecticide
Ammonium sodium phosphate $NaNH_4HPO_4$	Odorless crystals mp approx 80°		As a reagent for determination of Mg and Zn, and in blowpipe analysis; for standardizing uranium solutions; in the preparation of ammonium phosphate and ammonium molybdate stock solutions

(continued)

TABLE 4 (Continued)

PROPERTIES, TOXICITY, AND USES OF PHOSPHORUS AND SOME PHOSPHORUS COMPOUNDS

Compound	Properties	Toxicity	Uses
Ferric phosphate $FePO_4$	White, gray, or pink crystals		As food and feed supplement, particularly in bread enrichment; as fertilizer
Malathion $C_{10}H_{19}O_6PS_2$	Deep brown to yellow liquid	Produces symptoms similar to those for parathion. Malathion is, however, considered to be less toxic	As insecticide, particularly effective against pear psylla, red spider mites, and aphids; appears to be about 100 times less toxic to warm-blooded animals than parathion, about two to four times less toxic to insects. Vet use: ectoparasiticide for horn fly, lice in dairy cattle; horn fly, lice, ticks in beef cattle; lice, sheep ked ticks in sheep and goats; lice, sarcoptic mange in swine; lice, ticks, mites in poultry

APPENDIX

TABLE 5

COMPARATIVE ACUTE TOXICITY OF CHOLINERGIC ORGANOPHOSPHORUS INSECTICIDES IN NORMAL MALE AND FEMALE RATS[52]

Insecticide	Number of Rats		LD_{50}±SE* (mg/kg)	
	Male	Female	Male	Female
Phosdrin	35	35	3.2±0.3	1.2±0.1
Systox	35	60	3.9±0.1	1.4±0.1
Guthion	37	37	4.0±0.5	8.7±0.6
Di-Syston	35	32	6.7±0.6	2.1±0.1
Parathion	57	41	8.1±0.4	2.5±0.4
Methyl	52	44	9.3±0.8	7.0±0.6
OMPA	78	39	9.6±0.4	28.7±1.3
Co-Ral	36	45	14.7±1.4	7.5±0.4
EPN	70	44	23.9±1.1	7.3±0.5
Trithion	72	34	27.0±0.8	10.1±0.8
Delnav	75	32	33.2±2.2	17.2±0.9
Ethion	36	37	34.5±5.9	25.9±1.8
Polex	54	44	67.7±5.0	124.0±8.8
Ronnel	38	25	118.0±16.2	2822.6±137.4
Malathion	48	39	193.0±25.9	619.4±25.0

*SE=standard error.

APPENDIX

TABLE 6

COMPARATIVE TOXICITIES OF SOME PHOSPHORUS COMPOUNDS FOR EXPERIMENTAL ANIMALS

Toxic Substance	Concentration Value (ppm body wt)	Route[e]	Subject	Effect	Ref.
			Single Exposure		
p-Nitrophenyldiethyl thiophosphate (E.605)	3.0	iv	Rat, male	LD	1
"	"	"	"	"	"
isomer I	1.2	"	"	"	"
isomer II	0.5	"	"	"	"
p-Nitrophenyldiethyl phosphate (E.600)	0.4	"	"	"	"
p-Nitrophenyldiethyl thiophosphate	10.0	"	Rabbit	10ED[a]83	"
"	"	"	"	10ED8[b]	"
"	"	ip	"	11ED66	"
"	"	"	"	11ED9[b]	"
"	"	"	"	40ED81	"
"	"	"	"	40ED20[b]	"
4-Methyl-7-hydroxy-coumaryldiethyl thiophosphate (E.838)	15.0	iv	"	20ED94	"
"	"	"	"	20ED26[b]	"
"	"	"	"	30ED97	"
"	"	"	"	30ED0[b]	"
Dimethyl p-nitrophenyl thiophosphate	10.0	"	"	10ED67	"
"	"	"	"	10ED0[b]	"
"	"	"	"	40ED64	"
"	"	"	"	40ED8[b]	"
"	"	"	"	10ED80	"
"	"	"	"	10ED6[b]	"
"	15.0	"	"	10ED80	"
"	"	"	"	10ED6[b]	"
"	"	"	"	40ED65	"
"	"	"	"	40ED5[b]	"

(continued)

TABLE 6 (Continued)

COMPARATIVE TOXICITIES OF SOME PHOSPHORUS COMPOUNDS FOR EXPERIMENTAL ANIMALS

Toxic Substance	Concentration Value (ppm body wt)	Route[e]	Subject	Effect	Ref.
			Single Exposure		
Di-isopropyl p-nitrophenyl thiophosphate	15.0	iv	Rabbit	10ED34	1
"	"	"	"	10ED21[b]	"
Bis-dimethylaminofluorophosphine oxide	20.0	"	"	7ED88	"
"	"	"	"	7ED31[b]	"
"	"	"	"	20ED86	"
"	"	"	"	20ED0[b]	"
"	"	"	"	42ED89	"
"	"	"	"	42ED0[b]	"
p-Nitrophenyldiethyl thiophosphate (E.605)	$1.7X10^{-4}$	sol	Sheep[c]	ED_{50}[b]	"
isomer I	$2.5X10^{-7}$	"	"	"	"
isomer III	$2.8X10^{-8}$	"	"	"	"
p-Nitrophenyldiethyl phosphate (E.600)	$2.0X10^{-8}$	"	"	"	"
4-Methyl-7-hydroxy-phosphate	$5.0X10^{-9}$	"	"	"	"
bis-dimethylaminoglu-orophosphine oxide	$4.0X10^{-5}$	"	Rat[d]	min ED_{50}[b]	1
"	$7.0X10^{-2}$	"	"	"	"
"	$7.0X10^{-4}$	"	"	"	"
	mg/m^3		4 hours		
Phosphorus oxychloride	61.3	ih	Rat, female 20	LD_{50}	79
"	56.3	"	"		"

(Continued)

TABLE 6 (Continued)

COMPARATIVE TOXICITIES OF SOME PHOSPHORUS COMPOUNDS FOR EXPERIMENTAL ANIMALS

Toxic Substance	Concentration Value (Moles)	Route[e]	Subject	Effect	Ref.
			Single Exposure		
Phosphorus oxychloride	66.6	ih	Guinea pig male, 10	LD_{50}	1
"	52.4	"	"	"	"
Phosphorus trichloride	132.1	"	Rat, female 20	"	"
	152.4	"	"	"	"
Phosphorus trichloride (vapors/aerosols)	63.5	"	Guinea pig male, 10	"	"
"	132.0	"	"	"	"

[a]ED = Effective dose for inhibition of cholinesterase. The right-hand subscript indicates the percent inhibition of cholinesterase and the left-hand one showes time (min) after injection or of incubation.

[b]in vitro inhibition.

[c]Incubation at 37° C, sheep red cells.

[d]Brain, auxiliary glands, red cells, brain serum, and heart.

[e]iv = intravenous; ip = intraperitoneal; ih = inhalation; sol = solution

APPENDIX

TABLE 7

PRODUCTION OF PHOSPHORUS CHEMICALS[19,20]
(Thousand Short Tons)

Phosphorus Chemicals	1964[a]	1965[a]	1966[a]	1967[b]	1968[c]	1969[c]
Phosphoric acid, total	3,283	3,905	4,549	4,764	5,202	5,690
Phosphorus, elemental	504	550	566	583	620	652
Phosphorus oxychloride	27	27	31	33	34	36
Phosphorus pentasulfide	42	50	54	45	58	64
Potassium pyrophosphate	52	55	53	53	63	69
Calcium phosphate, basic	242	263	290	380	339	356
Sodium phosphate, tripoly	886	923	1,001	1,046	1,072	1,117
Potassium pyrophosphate	52	55	53	53	63	69

[a]U.S. Department of Commerce.

[b]Preliminary data.

[c]C & EN computer forecasts.

TABLE 8

GROWTH OF PHOSPHORIC ACID INDUSTRY IN UNITED STATES[2]
(ton/year 100% P_2O_5 basis

Year	Thermal process	Wet process
1941	108,494	131,521
1942	104,655	118,970
1943	103,418	127,248
1944	111,157	141,141
1945	132,149	132,599
1947	191,911	174,987
1948	211,578	220,828
1949	259,677	245,427
1950	295,073	299,152
1951	330,096	338,426
1952	357,622	388,908
1953	462,580	496,152
1954	506,774	631,252
1955	540,025	774,998
1956	570,235	811,770
1957	632,954	936,129
1958	657,372	1,033,205
1959	740,747	1,140,658
1960	762,531	1,324,695
1961	844,761	1,409,173
1962	869,805	1,576,976
1963	947,293	1,957,476
1964	1,007,624	2,275,418
1965	1,007,941[a]	2,837,119

[a]Subject to revision

TABLE 9

THERMAL-PROCESS PHOSPHORIC ACID ESTABLISHMENTS IN UNITED STATES[2]

State	Company	Location
Alabama	Tennessee Valley Authority	Wilson Dam
California	F M C Corporation	Newark
	Monsanto Company	Long Beach
	Stauffer Chemical Company	Richmond
	Stauffer Chemical Company	South Gate
Colorado	Colorado Fuel and Iron Corporation	Pueblo (not operating)
Georgia	Monsanto Company	Augusta
Idaho	El Paso Natural Gas Company	Georgetown (not operating)
Illinois	Monsanto Company	Sauget
	Stauffer Chemical Company	Chicago Heights
	Stauffer Chemical Company	South Chicago
Indiana	Hooker Chemical Company	Jeffersonville
	Mobil Chemical Company	Gary
Kansas	F M C Corporation	Lawrence
Massachusetts	Hooker Chemical Company	Adams
Michigan	Monsanto Company	Trenton
Montana	Stauffer Chemical Company	Butte
New Jersey	Agrigo Chemical Company	Carteret
	Continental Oil Company	Carteret
	F M C Corporation	Carteret
	Monsanto Company	Kearny
New York	Hooker Chemical Company	Niagara Falls (not operating)
Ohio	Mobil Chemical Company	Fernald
	Monsanto Company	Addyston

(continued)

TABLE 9 (Continued)

THERMAL-PROCESS PHOSPHORIC ACID ESTABLISHMENTS IN UNITED STATES[2]

State	Company	Location
Pennsylvania	Stauffer Chemical Company	Morrisville
South Carolina	Mobil Chemical Company	Charleston
Tennessee	Hooker Chemical Company	Columbia
	Stauffer Chemical Company	Nashville
Texas	Hooker Chemical Company	Dallas
Wyoming	F M C Corporation	Green River